June 1992
Volume 37, Number 6
ISSN 00989142-RECACP

RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
37TH YEAR—ESTABLISHED 1956

SPECIAL ISSUE
Conference on
Emergency Respiratory Care
Part 1

- The Respiratory Therapist's Role in Emergency Care
- Upper Airway Problems
- Emergency Management of Acute, Severe Asthma
- Diagnosis and Management of Massive Hemoptysis
- Neonatal and Pediatric Airway Emergencies
- Near-Drowning
- Smoke Inhalation Injury

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References 1 Data on file, HealthScan Products Inc., 2 Shapiro S, Hendler J, Ogirala R, et al An evaluation of the accuracy of Assess and MiniWright peak flowmeters *Chest* 99 358-362, 1991

ASSESS and ASSESS Low Range meet *National Asthma Education Program Technical Standards for Peak Flow Meters*, January, 1991. AA710001-0 10/91

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Cyclosporin in Chronic Severe Asthma (editorial)—Anonymous. Lancet 1992;339:338. (See related paper by Alexander et al below.)

Inhaled Glucocorticoid Therapy in Children: How Much Is Safe? (editorial)—AK Kamada, DP Parks, SJ Szefler. Pediatr Pulmonol 1992;12:71-72. (See related paper by Phillip et al below.)

Retinopathy of Prematurity (editorial). DL Phelps. N Engl J Med 1992;326:1078. (See related paper by Flynn et al abstracted on Page 497.)

Integrated Plasma Cortisol Concentration in Children with Asthma Receiving Long-Term Inhaled Corticosteroids—M Phillip, M Aviram, E Leiberman, Z Zadik, Y Giat, J Levy, et al. Pediatr Pulmonol 1992;12:84.

We assessed the effect of long-term therapy with inhaled beclomethasone dipropionate (BDP) on the pituitary-adrenal axis, by measuring the integrated concentration (IC) of plasma cortisol in 8 children with asthma (age 6-16 y) who regularly used inhaled BDP in doses ranging from 8 to 26.5 μg/kg (200-450 μg/day) for 6 months to 4 years. The control group included 6 chldren (age 6-16 y) who had the IC of plasma cortisol measured as part of an endocrinological evaluation and were found to be healthy. Cortisol concentration was measured in blood samples collected continuously over a 24-h period. Mean IC of plasma cortisol in the study group was significantly lower than in the healthy controls (mean ± SD, 4.9 ± 3.3 vs 9.1 ± 1.9 μg/mL; $p < 0.02$). Cortisol response to 0.25 mg ACTH (iv) was abnormal in 1 of the 8 BDP-treated patients. No correlation was found between IC of plasma cortisol and the BDP dose, severity of asthma, height percentile, or the Tanner stage. We conclude that long-term therapy, even with relatively conventional doses of inhaled BDP may cause reduction in the normal physiological secretion or cortisol. The clinical relevance of low IC of plasma cortisol is not clear, but it may reflect partial suppression of the pituitary-adrenal axis.

Trial of Cyclosporin in Corticosteroid-Dependent Chronic Severe Asthma—A Alexander, NC Barnes, AB Kay. Lancet 1992;339:324.

The treatment of chronic severe asthma is unsatisfactory for many patients. In a randomised, double-blind, placebo-controlled, crossover trial we have tested whether cyclosporin, which is thought to act primarily by inhibition of T lymphocyte activation improves lung function in corticosteroid-dependent asthmatics. After a 4-week run-in period, 33 patients with long-standing asthma (mean duration 27 years), and who had required continuous oral corticosteroids for a mean of 9.3 years, were randomised to receive either cyclosporin (initial dose 5 mg/kg/day) or placebo for 12 weeks, crossing over after a 2-week washout period. Mean baseline forced expiratory volume in 1 s (FEV_1) was 60.1% of the predicted value. 2 patients failed to complete the protocol and 1 withdrew because of hypertrichosis. Cyclosporin therapy resulted in a mean increase above placebo of 12.0% in morning peak expiratory flowrate (PEFR, $p < 0.004$) and 17.6% in FEV_1 ($p < 0.001$). The frequency of disease exacerbations requiring an increased prednisolone dose was reduced by 48% in patients on cyclosporin compared with placebo ($p < 0.02$). Diurnal variation in PEFR decreased by a mean of 27.6% ($p = 0.04$). Cyclosporin for 12 weeks was well tolerated by this group of chronic asthmatics, in whom the mean whole-blood trough concentration was 152 μg/L. These findings

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INDICATIONS AND USAGE

SURVANTA is indicated for prevention and treatment ("rescue") of Respiratory Distress Syndrome (RDS) (hyaline membrane disease) in premature infants SURVANTA significantly reduces the incidence of RDS, mortality due to RDS and air leak complications

Prevention

In premature infants less than 1250 g birth weight or with evidence of surfactant deficiency, give SURVANTA as soon as possible, preferably within 15 minutes of birth

Rescue

To treat infants with RDS confirmed by x-ray and requiring mechanical ventilation, give SURVANTA as soon as possible, preferably by 8 hours of age

CONTRAINDICATIONS

None known

WARNINGS

SURVANTA is intended for intratracheal use only

SURVANTA CAN RAPIDLY AFFECT OXYGENATION AND LUNG COMPLIANCE Therefore, its use should be restricted to a highly supervised clinical setting with immediate availability of clinicians experienced with intubation, ventilator management, and general care of premature infants. Infants receiving SURVANTA should be frequently monitored with arterial or transcutaneous measurement of systemic oxygen and carbon dioxide

DURING THE DOSING PROCEDURE, TRANSIENT EPISODES OF BRADYCARDIA AND DECREASED OXYGEN SATURATION HAVE BEEN REPORTED If these occur, stop the dosing procedure and initiate appropriate measures to alleviate the condition. After stabilization, resume the dosing procedure

PRECAUTIONS

General

Rales and moist breath sounds can occur transiently after administration. Endotracheal suctioning or other remedial action is not necessary unless clear-cut signs of airway obstruction are present

Increased probability of post-treatment nosocomial sepsis in SURVANTA-treated infants was observed in the controlled clinical trials (Table 3) The increased risk for sepsis among SURVANTA-treated infants was not associated with increased mortality among these infants The causative organisms were similar in treated and control infants There was no significant difference between groups in the rate of post-treatment infections other than sepsis

Use of SURVANTA in infants less than 600 g birth weight or greater than 1750 g birth weight has not been evaluated in controlled trials There is no controlled experience with use of SURVANTA in conjunction with experimental therapies for RDS (eg, high-frequency ventilation or extracorporeal membrane oxygenation)

No information is available on the effects of doses other than 100 mg phospholipids/kg more than four doses, dosing more frequently than every 6 hours, or administration after 48 hours of age

Carcinogenesis, Mutagenesis, Impairment of Fertility

Reproduction studies in animals have not been completed Mutagenicity studies were negative Carcinogenicity studies have not been performed with SURVANTA

ADVERSE REACTIONS

The most commonly reported adverse experiences were associated with the dosing procedure In the multiple-dose controlled clinical trials, transient bradycardia occurred with 11 9% of *doses*. Oxygen desaturation occurred with 9 8% of *doses*

Other reactions during the dosing procedure occurred with fewer than 1% of doses and included endotracheal tube reflux, pallor, vasoconstriction, hypotension, endotracheal tube blockage, hypertension, hypocarbia, hypercarbia, and apnea No deaths occurred during the dosing procedure, and all reactions resolved with symptomatic treatment

The occurrence of concurrent illnesses common in premature infants was evaluated in the controlled trials The rates in all controlled studies are in Table 3

TABLE 3

	All Controlled Studies		
Concurrent Event	**SURVANTA (%)**	**Control (%)**	***P*-Value[a]**
Patent ductus arteriosus	46 9	47 1	0 814
Intracranial hemorrhage	48 1	45 2	0 241
Severe intracranial hemorrhage	24 1	23 3	0 693
Pulmonary air leaks	10 9	24 7	< 0 001
Pulmonary interstitial emphysema	20 2	38 4	< 0 001
Necrotizing enterocolitis	6 1	5 3	0 427
Apnea	65 4	59 6	0 283
Severe apnea	46 1	42 5	0 114
Post-treatment sepsis	20 7	16 1	0 019
Post-treatment infection	10 2	9 1	0 345
Pulmonary hemorrhage	7 2	5 3	0 166

[a] *P*-value comparing groups in controlled studies

When all controlled studies were pooled, there was no difference in intracranial hemorrhage However, in one of the single-dose rescue studies and one of the multiple-dose prevention studies, the rate of intracranial hemorrhage was significantly higher in SURVANTA patients than control patients (63 3% *v* 30 8%, *P* = 0 001, and 48 8% *v* 34 2%, *P* = 0 047, respectively). The rate in a Treatment IND involving approximately 4400 infants was lower than in the controlled trials

In the controlled clinical trials, there was no effect of SURVANTA on results of common laboratory tests: white blood cell count and serum sodium, potassium, bilirubin, creatinine

More than 3700 pretreatment and post-treatment serum samples were tested by Western Blot immunoassay for antibodies to surfactant-associated proteins SP-B and SP-C No IgG or IgM antibodies were detected

Several other complications are known to occur in premature infants The following conditions were reported in the controlled clinical studies The rates of the complications were not different in treated and control infants, and none of the complications were attributed to SURVANTA

Respiratory lung consolidation, blood from the endotracheal tube, deterioration after weaning, respiratory decompensation, subglottic stenosis, paralyzed diaphragm, respiratory failure

Cardiovascular hypotension, hypertension, tachycardia, ventricular tachycardia, aortic thrombosis, cardiac failure, cardiorespiratory arrest, increased apical pulse, persistent fetal circulation, air embolism, total anomalous pulmonary venous return

Gastrointestinal abdominal distention, hemorrhage, intestinal perforations, volvulus, bowel infarct, feeding intolerance, hepatic failure, stress ulcer

Renal renal failure, hematuria

Hematologic coagulopathy, thrombocytopenia, disseminated intravascular coagulation

Central Nervous System seizures

Endocrine/Metabolic: adrenal hemorrhage, inappropriate ADH secretion, hyperphosphatemia

Musculoskeletal inguinal hernia

Systemic fever, deterioration

Follow-Up Evaluations

To date, no long-term complications or sequelae of SURVANTA therapy have been found

Single-Dose Studies

Six-month adjusted-age follow-up evaluations of 232 infants (115 treated) demonstrated no clinically important differences between treatment groups in pulmonary and neurologic sequelae, incidence or severity of retinopathy of prematurity, rehospitalizations, growth, or allergic manifestations

Multiple-Dose Studies

Six-month adjusted age follow-up evaluations have not been completed Preliminarily, in 605 (333 treated) of 916 surviving infants, there are trends for decreased cerebral palsy and need for supplemental oxygen in SURVANTA infants. Wheezing at the time of examination tended to be more frequent among SURVANTA infants, although there was no difference in bronchodilator therapy

Twelve-month follow-up data from the multiple-dose studies have been completed in 328 (171 treated) of 909 surviving infants. To date no significant differences between treatments have been found, although there is a trend toward less wheezing in SURVANTA infants in contrast to the six month results

OVERDOSAGE

Overdosage with SURVANTA has not been reported Based on animal data, overdosage might result in acute airway obstruction Treatment should be symptomatic and supportive

Rales and moist breath sounds can transiently occur after SURVANTA is given, and do not indicate overdosage Endotracheal suctioning or other remedial action is not required unless clear-cut signs of airway obstruction are present

HOW SUPPLIED

SURVANTA (beractant) Intratracheal Suspension is supplied in single-use glass vials containing 8 mL of SURVANTA (NDC 0074-1040-08) Each milliliter contains 25 mg of phospholipids (200 mg phospholipids/8 mL) suspended in 0 9% sodium chloride solution. The color is off-white to light brown

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provide further evidence of a role for activated T lymphocytes in the pathogenesis of asthma. Specific pharmacological targeting of this cell could form the basis of a novel approach to the treatment of asthma.

A Successful Computerized Protocol for Clinical Management of Pressure Control Inverse Ratio Ventilation in ARDS Patients—TD East, SH Böhm, CJ Wallace, TP Clemmer, LK Weaver, JF Orme, AH Morris. Chest 1992;101:697.

We have developed a computerized protocol that provides a systematic approach for management of pressure control-inverse ratio ventilation (PCIRV). The protocols were used for 1,466 h in 10 around-the-clock PCIRV evaluations on 7 patients with severe adult respiratory distress syndrome (ARDS). Patient therapy was controlled by protocol 95% of the time (1,396 of 1,466 h) and 90% of the protocol instructions (1,937 of 2,158) were followed by the clinical staff. Of the 221 protocol instructions, 88 (39%) not followed were due to invalid intrinsic PEEP ($PEEP_i$) measurements. Compared with preceding values during controlled positive pressure ventilation (CPPV), the expired minute ventilation was reduced by 27% during PCIRV while maintaining a pH that was not clinically different (mean difference in pH = 0.02). There was no difference in the P_{aO_2}, $PEEP_i$, or the F_{IO_2} between PCIRV and CPPV. The PEEP setting was reduced by 33% from 9 ± 0.05 to 6 ± 0.6 and the I-E ratio increased from 0.64 ± 0.04 to 2.3 ± 0.10. Peak airway pressure was reduced by 24% (from 59 ± 1.5 to 45 ± 0.6) and mean airway pressure increased by 27% (from 22 ± 0.8 to 28 ± 0.6) in PCIRV. Right atrial and pulmonary artery pressures were higher and cardiac output lower in PCIRV but blood pressure was unchanged. The success of this protocol has demonstrated the feasibility of using $PEEP_i$ as a primary control variable for oxygenation. This computerized PCIRV protocol should make the future use of PCIRV less mystifying, simpler, and more systematic.

A Cohort Study of Transcutaneous Oxygen Tension and the Incidence and Severity of Retinopathy of Prematurity—JT Flynn, E Bancalari, ES Snyder, RN Goldberg, W Feuer, J Cassady, et al. N Engl J Med 1992;326:1050.

BACKGROUND: Retinopathy of prematurity is a disease affecting the blood vessels of the retina in premature infants that may result in scarring, retinal detachment, and loss of vision. An association between this condition and the exposure of premature infants to supplemental oxygen has been postulated, but the relation between retinopathy of prematurity and blood oxgyen levels has not been defined. The purpose of this study of a cohort of preterm infants was to correlate the incidence and severity of retinopathy of prematurity with the duration of exposure to different ranges of oxygen tension as measured by transcutaneous monitoring (tcP_{O_2}). METHODS: 101 premature infants (birthweight 500-1,300 g) requiring supplemental oxygen had continuous monitoring of tcP_{O_2}. The number of hours during which the tcP_{O_2} was 80 torr or higher was tabulated for each infant during the first 4 weeks of life. RESULTS: There was a significant association between the amount of time that the tcP_{O_2} was ≥ 80 torr and the incidence and severity of retinopathy of prematurity. The odds ratio for each 12-hour period in which the tcP_{O_2} was ≥ 80 torr was 1.9 (95% confidence interval 1.2 to 3.0) after adjustment for the following factors: birthweight ≤ 1,300 g (odds ratio 2.3 [95% confidence interval 1.6 to 3.4]), 5-minute Apgar score of 7 or less (odds ratio 7.2 [95% confidence interval 2.5 to 21]), and exposure to inspired oxygen at a fractional concentration ≥ 0.4 (odds ratio 1.0 [95 percent confidence interval 0.97 to 1.05]). The association was stronger for tcP_{O_2} values of ≥ 80 torr occurring from the second through the fourth week of life; during this period, the adjusted odds ratio for a 12-hour period of such exposure was 3.1 (95% confidence interval 1.6 to 6.1). CONCLUSIONS: This study supports an association between the incidence and severity of retinopathy of prematurity and the duration of exposure to arterial oxygen levels of 80 torr or higher, measured transcutaneously.

An Economic Evaluation of Asthma in the United States—KB Weiss, PJ Gergen, TA Hodgson. N Engl J Med 1992;326:862.

BACKGROUND: Asthma is a common chronic illness. Recently, increases in morbidity and mortality due to this disease have been reported. We studied the distribution of health care resources used for asthma in order to lay the groundwork for further policy decisions aimed at reducing the economic burden of this disorder. METHODS: Estimates of direct medical expenditures and indirect costs (in 1985 dollars) were derived from data available from the National Center for Health Statistics. These cost estimates were projected to 1990 dollars. RESULTS: The cost of illness related to asthma in 1990 was estimated to be $6.2 billion. Inpatient hospital services represented the largest single direct medical expenditure for this chronic condition, approaching $1.6 billion. The value of reduced productivity due to loss of school days represented the largest single indirect cost, approaching $1 billion in 1990. Although asthma is

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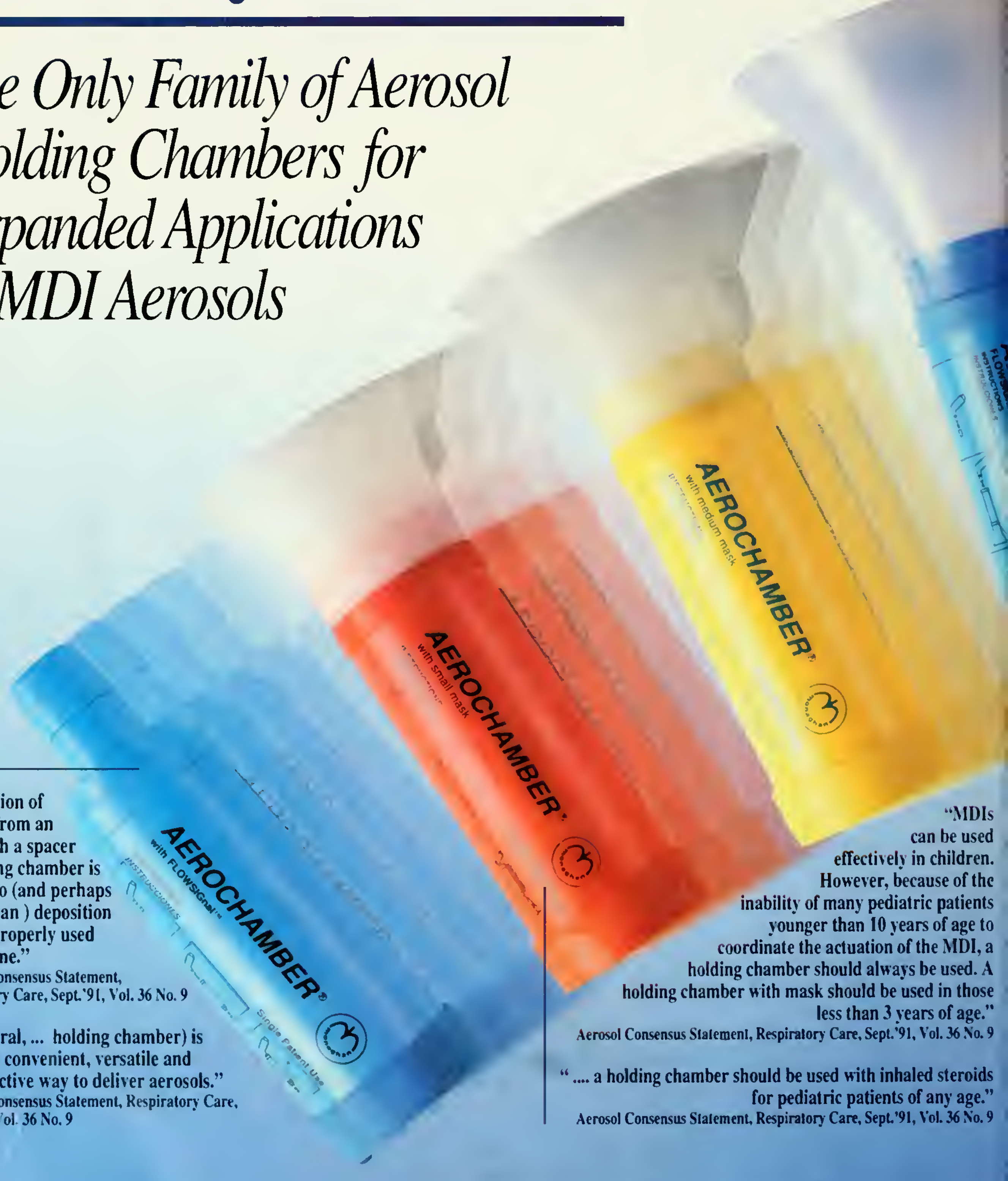

"Deposition of aerosol from an MDI with a spacer or holding chamber is similar to (and perhaps better than) deposition from a properly used MDI alone."
Aerosol Consensus Statement, Respiratory Care, Sept.'91, Vol. 36 No. 9

"In general, ... holding chamber) is the most convenient, versatile and cost-effective way to deliver aerosols."
Aerosol Consensus Statement, Respiratory Care, Sept.'91, Vol. 36 No. 9

"MDIs can be used effectively in children. However, because of the inability of many pediatric patients younger than 10 years of age to coordinate the actuation of the MDI, a holding chamber should always be used. A holding chamber with mask should be used in those less than 3 years of age."
Aerosol Consensus Statement, Respiratory Care, Sept.'91, Vol. 36 No. 9

" .... a holding chamber should be used with inhaled steroids for pediatric patients of any age."
Aerosol Consensus Statement, Respiratory Care, Sept.'91, Vol. 36 No. 9

The Aerosol Consensus Statement of the AARC, the NIH expert panel and practitioners of respiratory care have established new *community standards* for the use of Metered Dose Inhaler aerosol treatments and therapy.

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"This study has demonstrated that a MDI plus aerosol holding chamber delivers a nearly fivefold greater dose of aerosolized drug to the lungs in comparison with a jet nebulizer in patients receiving mechanical ventilation."
H.D. Fuller, M.B. Dolovich, G. Posmituck, W. Wong Pack, and M.T. Newhouse

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J. Grigg, et al Hammersmith Hospital, London, England

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often considered to be a mild chronic illness treatable with ambulatory care, we found that 43% of its economic impact was associated with emergency room use, hospitalization, and death. Nearly two thirds of the visits for ambulatory care were to physicians in three primary care specialties—pediatrics, family medicine or general practice, and internal medicine. CONCLUSIONS: Potential reductions in the costs related to asthma in the United States may be identified through a closer examination of the effectiveness of care associated with each category of cost. Future health policy efforts to improve the effectiveness of primary care interventions for asthma in the ambulatory setting may reduce the costs of this common illness.

Validity of Pulse Oximetry during Exercise in Elite Endurance Athletes—D Martin, S Powers, M Cicale, N Collop, D Huang, D Criswell. J Appl Physiol 1992;72(2):455.

Eleven highly trained male cyclists [maximal aerobic power ($\dot{V}_{O_{2max}}$) = 70.6 ± 4.2 mL · kg^{-1} · min^{-1}] performed both high intensity constant load (90-95% $\dot{V}_{O_{2max}}$) and incremental cycle exercise tests with arterial blood sampling to evaluate the accuracy of pulse oximeter estimates ($\%S_{pO_2}$) of arterial oxyhemoglobin fraction of total hemoglobin ($\%HbO_2$). Three subjects also performed an incremental exercise test in hypoxic conditions (inspired partial pressure of O_2 = 89, 93, or 100 torr). Arterial $\%HbO_2$ was determined via CO-oximetry and ranged from 72 to 99%. Three Ohmeda 3740 pulse oximeters were used to estimate $\%HbO_2$, one on each ear lobe and a finger probe. The finger probe tended to provide the best estimate of $\%HbO_2$ during exercise: the mean $\%S_{pO_2}$-$\%HbO_2$ difference for 232 exercise observations was 0.52 ± 1.36% (SD). Finger probe $\%S_{pO_2}$ and $\%HbO_2$ were highly correlated ($r = 0.98$; standard error of the estimate, SEE = 1.32%; $p < 0.0001$). The accuracy of pulse oximeters has been questioned during high-intensity exercise. When aerobic power was ≥ 81% of $\dot{V}_{O_{2max}}$ ($n = 75$), the finger probe's mean error was −0.01 ± 1.40%. Finger probe $\%S_{pO_2}$ and $\%HbO_2$ were highly correlated ($r = 0.97$, SEE = 1.32%, $p < 0.0001$). These results indicate that this pulse oximeter is a valid predictor of $\%HbO_2$ in elite athletes during cycle exercise.

Transcutaneous Oxygen and Carbon Dioxide during the First Half Year of Life in Premature and Normal Term Infants—T Haoppenbrouwers, JE Hodgman, K Arakawa, M Durand, LA Cabal. Pediatr Res 1992;31:73.

Repetitive polysomnograms were recorded from a total of 33 infants, 19 healthy preterm infants, and 14 term controls between 40-wk-postconceptional age and 6 mo of age. These nighttime recordings lasted 2-4 h, except at 52 wk in preterm infants and at 3 mo of age in term infants when an overnight 12-h recording was performed. Minute-by-minute values of transcutaneous P_{O_2} (P_{tcO_2}) and transcutaneous P_{CO_2} (P_{tcCO_2}) levels and variability during the awake state, active sleep, and quiet sleep were obtained through computer analyses of the polygraphic data. The results from preterm infants at corrected postconceptional age could not be differentiated from those of control infants. P_{tcO_2} levels rose between 40 wk and 3 mo, and P_{tcCO_2} levels declined. Sleep states modulated only the variability of P_{tcO_2}, not the level; in contrast, state modulation was seen in both variability and level of P_{tcCO_2} throughout the age span studied. During sleep the number of transient declines in P_{tcO_2} > 2.03 kPa (15 torr) decreased with advancing age. Hypercapnic P_{tcCO_2} values decreased with age as well, but their prevalence in healthy, young infants suggests the need for re-evaluation of criteria for hypercapnia based on transcutaneous measurements. The data demonstrate that ventilatory regulation continues to undergo changes between 1 and 3 mo, the age of highest risk for sudden infant death syndrome.

Deposition of Aerosolized Pentamidine and Failure of Pneumocystis Prophylaxis—GC Smaldone, G Dickinson, E Marcial, E Young, J Seymour. Chest 1992;101:82.

OBJECTIVE: To determine if outcome of *Pneumocystis carinii* prophylaxis is related to total lung dose of aerosolized pentamidine. SETTING: AIDS treatment centers at a VA and University Hospital. PATIENTS: 58 HIV-infected patients receiving *P carinii* prophylaxis with aerosolized pentamidine using a nebulizer (CIS-US AeroTech II) were followed up over a 90-week period. Treatment consisted of 60 to 90 mg every 2 weeks. MEASUREMENTS: In all patients, deposition of pentamidine aerosol was measured using a radioaerosol filter technique. Factors thought to be important in deposition, nebulizer output, and breathing pattern were also measured. Six months later, repeated deposition studies were performed in 20 patients. Pentamidine dose to the lung was related to occurrence of *P carinii* pneumonia and correlated with nebulizer function and breathing parameters. Outcome was assessed in terms of pentamidine deposition and patient characteristics, including demographic, immunologic, physiologic, and medical variables. RESULTS: 10 patients (17.2%) had development of *P carinii* pneumonia. However, pentamidine deposited in the failures (8.18 ± 4.74 mg) was no different

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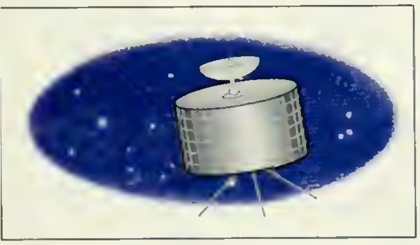

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Aerosol Administration

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Dean Hess, MEd, RRT and David J. Pierson, MD—Know the basic principles underlying the therapeutic use of drugs by the aerosol route as well as the differences in efficacy and use of metered-dose inhalers vs. nebulizers. Learn how to administer and assess the effects of bronchodilators during mechanical ventilation and the techniques for correct use of aerosol devices by patients.

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November 17, 1992, 12:30-2:00 p.m. Eastern Time

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In Part II, you will learn detailed information about the control of ventilation in terms of switching from inspiration to expiration. You will also be introduced to the common drive mechanisms used in various mechanical devices.
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Part III explores the output waveforms that ventilators produce and their clinical implications. Instruction is also given on the various modes of ventilation in terms of the specific combinations of control characteristics that make them unique along with ventilator performance testing and the practical application of ventilator classification in everyday communication.
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Microbiology for Respiratory Therapy: A Review of Microbial Growth and Cross-Contamination
Provides you with an overview of some important aspects of microbiology in respiratory care. Includes four main sections: groups and characteristics of microbes, requirements for microbial growth, cross-contamination, and prevention of transmission.
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Patient Evaluation

Pulmonary Function Assessment I: Basic Screening Studies
Helps you understand the results of simple spirometry tests and gives the definition of obstructive and restrictive pulmonary disorders and descriptions of the basic pathology involved in each. It further covers the four reasons for simple spirometry tests, the common parameters of lung function measured by simple spirometry, and helps you identify the results from spirometry indicative of obstructive and restrictive pulmonary disorders.
Item PE3 — $8 Ea

Pulmonary Function Assessment II: Bedside Studies
This study unit is designed to familiarize you with the use and interpretation of bedside pulmonary function tests. You will learn how pulmonary function test results can be used to assess ventilatory ability and which tests are commonly used. Also, learn what measuring instrument is required for each test, how the test is performed, and what is significant about the test results.
Item PE4 — $7 Ea

Pulmonary Function Assessment III: Lung Volume Determination and Closing Volume Studies
This material is designed to help you recall the eight subdivisions of total lung volume and their individual characteristics in health and disease. Also helps you understand the measurement of lung volume by nitrogen washout, helium dilution, and closing volume techniques.
Item PE5 — $6 Ea

Body Plethysmography
Explains the theory of body plethysmography and its use in the measurement of FRC and airway resistance. Defines Boyle's Law, thoracic volume, airway resistance, and its normal values. Also describes the measurement of airway resistance using body plethysmography.
Item PE6 — $9 Ea

Sources of Error in the Determination of Blood Gas Values and pH
This study package will familiarize you with the errors that may occur in the analysis of blood gases and pH. These are often the most important laboratory data used in the diagnosis and treatment of pulmonary disease, and errors in these values can result in deleterious effects on patient care.
Item PE7 — $10 Ea

Temperature Adjustment of Blood Gases and pH
The principal goal of this study package is to teach you the effects of abnormal body temperature on blood gas and pH values. Blood gas values and pH are determined at 37 degrees Celsius, but if a patient's temperature is abnormal, the in-vivo blood gas and pH values will be different from those reported by the blood gas laboratory. This package will teach you how to adjust these values.
Item PE8 — $10 Ea

Measurement of Static Compliances and Dynamic Characteristics Curves During Mechanical Ventilation
Upon completion of this package, you will be able to compute and record static compliance curves and dynamic characteristics. You will also be able to describe the procedure of obtaining pressure-volume measurements and be able to interpret the compliance characteristics measurements.
Item PE9 — $7 Ea

Arterial Blood Gas Interpretation
Teaches you an understanding of ABG interpretation in order that the therapy prescribed by the physician can be administered in a knowledgeable manner. A systematic method will be described that allows you to correctly classify the acid/base dysfunction and to relate the diagnosis concisely and coherently to other members of the health care team.
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Chest Tubes and Pleural Drainage
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Tracheal Intubation I: Upper Airway Anatomy and Goals of Intubation
After completing this HSP, you will understand the rationale for tracheal intubation and be able to identify the important landmarks of upper airway anatomy.
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Tracheal Intubation II: Routes of Intubation
Describes the four routes of tracheal intubations and some advantages and hazards of each. Also presents the process for selecting the most suitable route in a given situation.
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Tracheal Intubation III: Equipment Procedures for Intubation
Covers the selection of the proper equipment necessary to perform endotracheal intubation and to ensure that they are in working order.
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Respiratory Management of Neuromuscular Crisis
This HSP will be helpful in the respiratory management of patients with ventilatory failure caused by a neuromuscular disorder. It will provide you with a basic understanding of how neuromuscular conditions lead to ventilatory insufficiency and what special considerations must be taken when working with these types of patients.
Item CP7 — $7 Ea

Respiratory Management of Flail Chest
This study package helps you increase your understanding of the pathophysiology of flail chest and the respiratory management of patients who have sustained chest wall trauma that results in a flail chest.
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Respiratory Management of Head Trauma
This HSP teaches you to identify five physical signs indicative of head trauma and explains the development of respiratory failure secondary to trauma. Also discusses airway management, drug therapy, ventilator parameters, acid-base status, and measures to be taken to maintain the appropriate pH, P_{aCO_2} and P_{aO_2}.
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Smoking Cessation

Nicotine Dependency Evaluation and Treatment
This HSP will help you understand the physiologic effects of nicotine, tests, and questions used to evaluate dependency. It will give you an understanding of nicotine and how it affects the chemistry of the brain, how it can be measured in bodily fluids, and the value of using self-tests to determine addiction levels. This HSP also teaches the importance of nicotine replacement and how it can be enhanced with behavioral counseling.
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Bedside Counseling of the Hospitalized Smoker
An HSP to prepare you for the role of smoking cessation counselor to hospital inpatients. Hospitalization presents a unique opportunity to assist patients to quit smoking. This HSP helps you understand the role of the bedside nicotine-dependence counselor and how to assess the inpatient smoker. You will be able to help patients cope with nicotine withdrawal, increase motivation for permanent cessation, and be able to understand factors contributing to smoking relapse.
Item SC2 — $10 Ea

than deposition in protected patients (6.39 ± 3.07 mg, p = NS). Most of the variability in deposition was accounted for by variability in nebulizer output (r = 0.919, p < 0.001). Deposition did not significantly correlate with any of the measured breathing parameters. Serial deposition measurements were not significantly different by paired analysis. The incidence of *P carinii* pneumonia did not correlate with any measured patient characteristic. CONCLUSIONS: Failure of aerosolized pentamidine prophylaxis is not related to total lung dose of pentamidine. Other factors such as inadequate microscopic deposition between alveoli, pentamidine clearance, or drug resistance may be important. In HIV-infected patients, interpatient variability in aerosol deposition can be reduced by reducing variability in nebulizer output rather than control of breathing pattern.

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Interlaboratory and Intralaboratory Variability in Pulmonary Function Testing: A 13-Year Study Using a Normal Biologic Control—KM Kangalee, RT Abboud. Chest 1992;101:88.

The purpose of this study was to document the precision with which measurements can be made on a normal subject as a "biologic control" when comparisons are made both on an intralaboratory and interlaboratory basis. One individual had a total of about 300 routine pulmonary function measurements in 22 laboratories during a 13-year period. In this study, significant variation was found in interlaboratory testing, while less variation was found in intralaboratory measurements. This technique of periodic testing of normal individuals as biologic controls is useful for documenting reproducibility and precision; evaluation of accuracy will require a check of the physical calibration of the equipment. The testing (on at least a monthly basis) of a normal nonsmoking subject as a biologic control is recommended as an integral component of quality assurance in the pulmonary function laboratory.

Respiratory Tract Infection Complicating Transtracheal Oxygen Therapy—JI Couser Jr, BJ Make. Chest 1992;101:273.

Transtracheal oxygen is generally well tolerated in patients with chronic hypoxemia. Minor complications are common, but there are few reports of serious respiratory tract infections associated with transtracheal oxygen therapy. We describe 4 patients with interstitial lung disease who had frequent lower respiratory tract infections requiring hospitalization after initiation of transtracheal oxygen therapy.

Hospital and Posthospital Survival in Patients Mechanically Ventilated for More than 29 Days—DR Gracey, JM Naessens, I Krishan, HM Marsh. Chest 1992;101:211.

Experience with prolonged mechanical ventilation has improved over recent years. Retrospective analysis of the records of 104 patients older than 16 years of age who were mechanically ventilated for more than 29 days over a 29-month period from May 1986 to October 1988 revealed the following findings. The mean patient age was 66.3 ± 15.7 years (SD). The mean number of in-hospital ventilator days was 59.9 ± 36.7 days (range 29 to 247 days). The mean number of days of oral or nasal endotracheal intubation prior to tracheostomy (96 patients) was 21.5 ± 14.2 days. The mean length of hospital stay for the 104 patients was 79.9 ± 45.4 days. The majority of the 104 patients (82.6%) were surgical patients. Nine patients left the hospital receiving extended mechanical ventilation. Mortality was highest in multiple organ system failure and lowest among the trauma patients. The total days of mechanical ventilation did not appear to be related to mortality if patients older than 16 years survived for 7 days. Postdischarge survival of the 53 of 60 patients who survived and whom we were able to contact was 67% at 1 year and 56% at 3 years.

Improvement in Exercise Capacity after Nocturnal Positive Pressure Ventilation and Tracheostomy in a Postpoliomyelitis Patient—CA Vaz Fragoso, RM Kacmarek, DM Systrom. Chest 1992;101:254.

Progressive neuromuscular symptoms years after recovery from acute paralytic poliomyelitis have been termed the PPS. We describe a 52-year-old man who contracted poliomyelitis at age 9 years who fully

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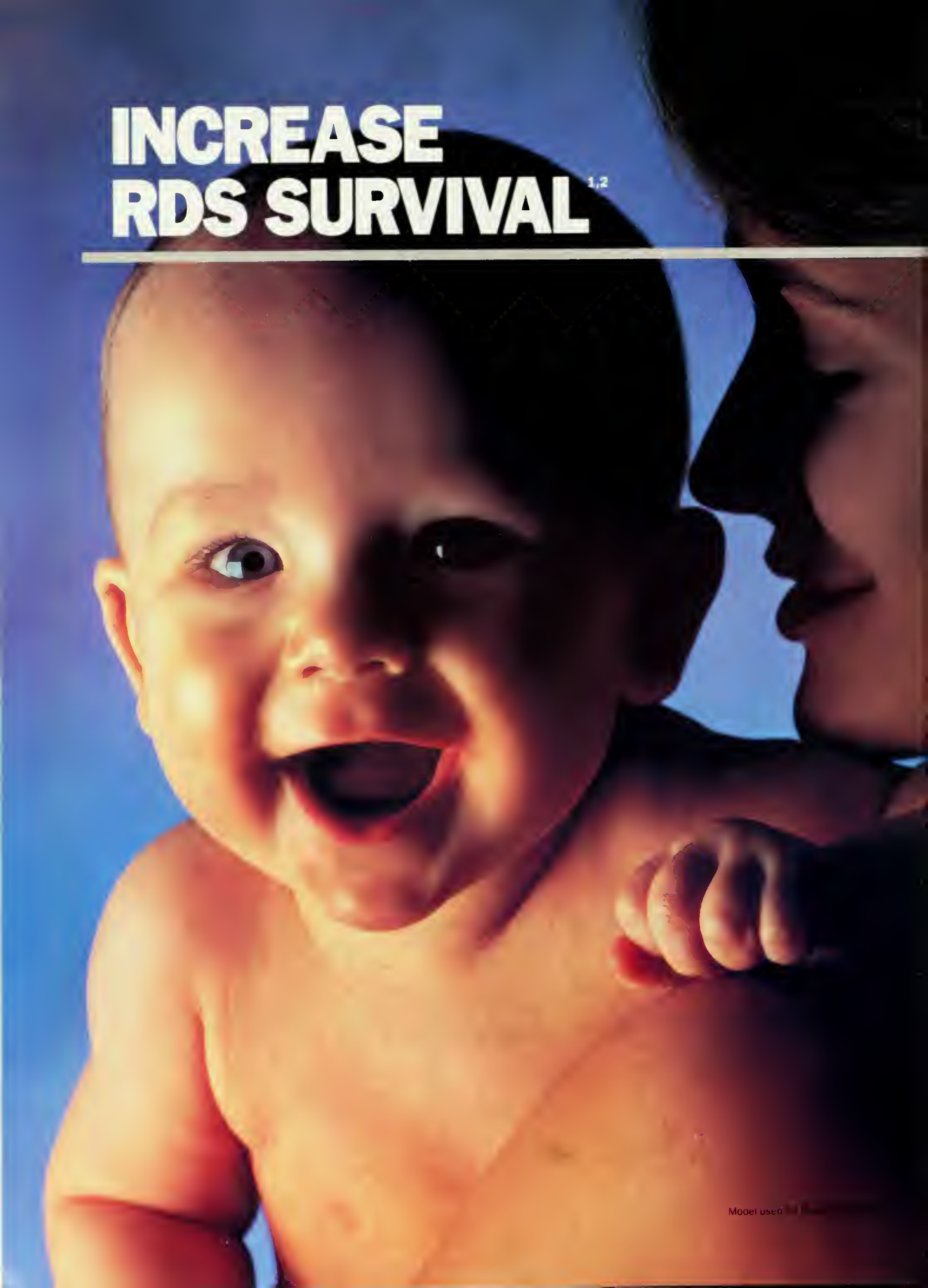

INCREASE RDS SURVIVAL[1,2]

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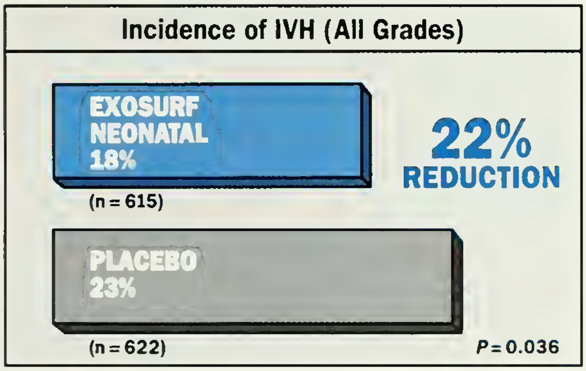
Significant IVH Reduction in Infants ≥1250 g

Despite successes in improving the survival of infants with RDS, intraventricular hemorrhage (IVH) remains a serious problem.[3-5] However, in a recently reported placebo-controlled rescue trial in infants ≥1250 g, EXOSURF Neonatal actually *reduced* the incidence of IVH.[2] In trials of smaller infants (<1250 g), EXOSURF Neonatal has never been observed to significantly increase IVH.



Two-dose treatment. Incidence of IVH in infants weighing 1250 g or more.
Adapted from Long et al[2](p1700)

Significant BPD Reduction in Infants ≥1250 g

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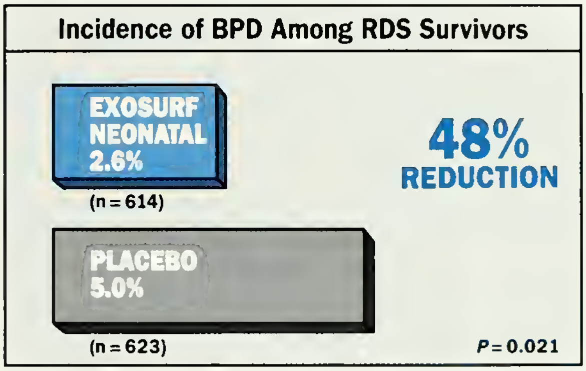

Two-dose treatment. Incidence of BPD in 28-day survivors following RDS in infants weighing 1250 g or more.
Adapted from Long et al[2](p1699)

No Increase in Sepsis

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INDICATIONS AND USAGE: Exosurf Neonatal is indicated for 1. **Prophylactic** treatment of infants with birth weights of less than 1350 grams who are at risk of developing RDS (see PRECAUTIONS), 2 **Prophylactic** treatment of infants with birth weights greater than 1350 grams who have evidence of pulmonary immaturity, and 3 **Rescue** treatment of infants who have developed RDS

CONTRAINDICATIONS: There are no known contraindications to treatment with Exosurf Neonatal

WARNINGS: **Intratracheal Administration Only:** Exosurf Neonatal should be administered only by instillation into the trachea (see DOSAGE AND ADMINISTRATION). **General:** The use of Exosurf Neonatal requires expert clinical care by experienced neonatologists and other clinicians who are accomplished at neonatal intubation and ventilatory management Adequate personnel, facilities, equipment, and medications are required to optimize perinatal outcome in premature infants Vigilant clinical attention should be given to all infants prior to, during, and after administration of Exosurf Neonatal **Acute Effects:** Exosurf Neonatal can rapidly affect oxygenation and lung compliance **Lung Compliance:** If chest expansion improves substantially after dosing, peak ventilator inspiratory pressures should be reduced immediately, without waiting for confirmation of respiratory improvement by blood gas assessment Failure to reduce inspiratory ventilator pressures rapidly in such instances can result in lung overdistention and fatal pulmonary air leak **Hyperoxia:** If the infant becomes pink and transcutaneous oxygen saturation is in excess of 95%, FiO_2 should be reduced in small but repeated steps (until saturation is 90 to 95%) without waiting for confirmation of elevated arterial pO_2 by blood gas assessment Failure to reduce FiO_2 in such instances can result in hyperoxia **Hypocarbia:** If arterial or transcutaneous CO_2 measurements are <30 torr, the ventilator rate should be reduced at once. Failure to reduce ventilator rates in such instances can result in marked hypocarbia, which is known to reduce brain blood flow **Pulmonary Hemorrhage:** In the single study conducted in infants weighing <700 grams at birth, the incidence of pulmonary hemorrhage (10% vs 2% in the placebo group) was significantly increased in the Exosurf Neonatal group. None of the five studies involving infants with birth weights >700 grams showed a significant increase in pulmonary hemorrhage in the Exosurf Neonatal group In a cross-study analysis of these five studies, fatal pulmonary hemorrhage occurred in three infants, two in the Exosurf Neonatal group and one in the placebo group Mortality from all causes among infants who developed pulmonary hemorrhage was 43% in the placebo group and 37% in the Exosurf Neonatal group. Pulmonary hemorrhage in both Exosurf Neonatal and placebo infants was more frequent in infants who were younger, smaller, male, or who had a patent ductus arteriosus. Pulmonary hemorrhage typically occurred in the first 2 days of life in both treatment groups **Mucous Plugs:** Infants whose ventilation becomes markedly impaired during or shortly after dosing may have mucous plugging of the endotracheal tube, particularly if pulmonary secretions were prominent prior to drug administration. Suctioning of all infants prior to dosing may lessen the chance of mucous plugs obstructing the endotracheal tube. If endotracheal tube obstruction from such plugs is suspected, and suctioning is unsuccessful in removing the obstruction, the blocked endotracheal tube should be replaced immediately

PRECAUTIONS: **General:** In the controlled clinical studies, infants known prenatally or postnatally to have major congenital anomalies, or who were suspected of having congenital infection, were excluded from entry However, these disorders cannot be recognized early in life in all cases, and a few infants with these conditions were entered The benefits of Exosurf Neonatal in the affected infants who received drug appeared to be similar to the benefits observed in infants without anomalies or occult infection **Prophylactic Treatment—Infants <700 Grams:** In infants weighing 500 to 700 grams, a single prophylactic dose of Exosurf Neonatal significantly improved FiO_2 and ventilator settings, reduced pneumothorax and reduced death from RDS, but increased pulmonary hemorrhage (see WARNINGS) Overall mortality did not differ significantly between the placebo and Exosurf Neonatal groups Data on multiple doses in infants in this weight class are not yet available **Rescue Treatment—Number of Doses:** A small number of infants with RDS have received more than two doses of Exosurf Neonatal as rescue treatment Definitive data on the safety and efficacy of these additional doses are not available **Carcinogenesis, Mutagenesis, Impairment of Fertility:** Exosurf Neonatal at concentrations up to 10,000 μg/plate was not mutagenic in the Ames Salmonella assay Long-term studies have not been performed in animals to evaluate the carcinogenic potential of Exosurf Neonatal The effects of Exosurf Neonatal on fertility have not been studied

ADVERSE REACTIONS:

General: Premature birth is associated with a high incidence of morbidity and mortality Despite significant reductions in overall mortality associated with Exosurf Neonatal, some infants who received Exosurf Neonatal developed severe complications and either survived with permanent handicaps or died In controlled clinical studies evaluating the safety and efficacy of Exosurf Neonatal, numerous safety assessments were made In infants receiving Exosurf Neonatal, pulmonary hemorrhage apnea and use of methylxanthines were increased A number of other adverse events were significantly reduced in the Exosurf Neonatal group, particularly various forms of pulmonary air leak and use of pancuronium ***Reflux*** Reflux of Exosurf Neonatal into the endotracheal tube during dosing has been observed and may be associated with rapid drug administration If reflux occurs, drug administration should be halted and, if necessary peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O until the endotracheal tube clears ***>20% Drop in Transcutaneous Oxygen Saturation:*** If transcutaneous oxygen saturation declines during dosing drug administration should be halted and, if necessary, peak inspiratory pressure on the ventilator should be increased by 4 to 5 cm H_2O for 1 to 2 minutes In addition, increases of FiO_2 may be required for 1 to 2 minutes.

DOSAGE AND ADMINISTRATION: **Preparation of Suspension:** Exosurf Neonatal is best reconstituted immediately before use because it does not contain antibacterial preservatives However, the reconstituted suspension is chemically and physically stable when stored at 2° to 30°C (36° to 86°F) for up to 12 hours following reconstitution Solutions containing buffers or preservatives should not be used for reconstitution. **Do Not Use Bacteriostatic Water for Injection, USP.** Each vial of Exosurf Neonatal should be reconstituted only with **8 mL** of the accompanying diluent (preservative-free Sterile Water for Injection) **Dosage: Accurate determination of weight at birth is the key to accurate dosing.** ***Prophylactic Treatment:*** The first dose of Exosurf Neonatal should be administered as a single 5 mL/kg dose as soon as possible after birth. Second and third doses should be administered approximately 12 and 24 hours later to all infants who remain on mechanical ventilation at those times ***Rescue Treatment.*** Exosurf Neonatal should be administered in two 5 mL/kg doses. The initial dose should be administered as soon as possible after the diagnosis of RDS is confirmed The second dose should be administered approximately 12 hours following the first dose, provided the infant remains on mechanical ventilation **Use of Special Endotracheal Tube Adapter:** With each vial of Exosurf Neonatal for Intratracheal Suspension, five different sized endotracheal tube adapters each with a special right angle Luer®-lock sideport are supplied The adapters are clean but not sterile **Administration:** The infant should be suctioned prior to administration of Exosurf Neonatal. Exosurf Neonatal suspension is administered via the sideport on the special endotracheal tube adapter **WITHOUT INTERRUPTING MECHANICAL VENTILATION.** Each Exosurf Neonatal dose is administered in two 2 5 mL/kg half-doses Each half-dose is instilled slowly over 1 to 2 minutes (30 to 50 mechanical breaths) in small bursts timed with inspiration After the first 2.5 mL/kg half-dose is administered in the midline position, the infant's head and torso are turned 45° to the **right** for 30 seconds while mechanical ventilation is continued After the infant is returned to the midline position, the second 2 5 mL/kg half-dose is given in an identical fashion over another 1 to 2 minutes The infant's head and torso are then turned 45° to the **left** for 30 seconds while mechanical ventilation is continued, and the infant is then turned back to the midline position These maneuvers allow gravity to assist in the distribution of Exosurf Neonatal in the lungs During dosing heart rate, color, chest expansion, facial expressions, the oximeter, and the endotracheal tube patency and position should be monitored **Suctioning should not be performed for two hours after Exosurf Neonatal is administered, except when dictated by clinical necessity.**

HOW SUPPLIED: Exosurf Neonatal for Intratracheal Suspension is supplied in a carton containing one 10 mL vial of Exosurf Neonatal for Intratracheal Suspension, one 10 mL vial of Sterile Water for Injection, and five endotracheal tube adapters (2 5, 3 0, 3 5, 4 0, and 4 5 mm I.D.). (NDC 0081-0207-01). Store Exosurf Neonatal for Intratracheal Suspension at 15° to 30°C (59° to 86°F) in a dry place

EDUCATIONAL MATERIAL: A videotape on dosing is available from your Burroughs Wellcome Co representative. This videotape demonstrates techniques for safe administration of Exosurf Neonatal and should be viewed by health care professionals who will administer the drug

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Copr ©1992 Burroughs Wellcome Co All rights reserved EX-YO3216 recovered and 33 years later developed progressive dyspnea. Neurologic evaluation revealed bilateral paralysis of the vocal cords, generalized weakness, and accentuated mouth occlusion pressure and ventilatory responses to hypercapnic, hyperoxic breathing. An EMG and muscle biopsy showed changes consistent with acute and chronic denervation. Cardiopulmonary exercise evaluation demonstrated a pulmonary mechanical limit with excessive ventilation relative to CO_2 output. Tracheostomy and nocturnal positive pressure ventilation resulted in increased respiratory muscle strength, normalization of ventilatory drive and marked improvement in exercise capacity.

Utility of the Peak Expiratory Flow Rate in the Differentiation of Acute Dyspnea: Cardiac vs Pulmonary Origin—RM McNamara, DJ Cionni. Chest 1992;101:129.

This study examined the utility of a peak expiratory flow rate (PEFR) measurement in the differentiation of acute moderate to severe dyspnea secondary to congestive heart failure or chronic lung disease. A PEFR was determined in 41 episodes of acute respiratory distress in 40 patients prior to emergency department therapy. The mean PEFR ± SD for the congestive heart failure group ($n = 18$) was 224 ± 82 L/min, which was significantly higher ($p < 0.001$) than that of the chronic lung disease group ($n = 23$), which had a mean PEFR of 108 ± 49 L/min. No single cutoff value allowed 100% accurate classification, but the results suggest that the PEFR may be a useful adjunctive tool in the differentiation of acute dyspnea of cardiac vs pulmonary origin.

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The Effects on Survival of Early Treatment of Human Immunodeficiency Virus Infection— NMH Graham, SL Zeger, LP Park, SH Vermund, R Detels, CR Rinaldo, JP Phair. N Engl J Med 1992;326:1037.

BACKGROUND: Zidovudine has been shown to prolong survival in patients with the acquired immunodeficiency syndrome (AIDS) and, in persons with human immunodeficiency virus (HIV) infection but not AIDS, to delay the progression to AIDS. However, it is still uncertain whether treatment before the development of AIDS prolongs survival. METHODS: We analyzed data from a cohort of 2,162 high-risk men who were already seropositive for HIV Type 1 (HIV-1) and 406 men who seroconverted from October 1986 through April 1991. There were 306 deaths. The probabilities of death were compared among men at similar stages of disease who began zidovudine therapy before the diagnosis of AIDS and among those who did not. Relative risks of death were calculated for each of five initial disease states on the basis of CD4+ cell counts and clinical symptoms and signs appearing over follow-up periods of 6, 12, 18, and 24 months. Adjustments were also made for the use of prophylaxis against *Pneumocystis carinii* pneumonia (PCP). RESULTS: After we controlled for CD4+ cell count and symptoms, the use of zidovudine with or without PCP prophylaxis before the development of AIDS significantly reduced mortality in all follow-up periods. The relative risks of death were 0.43 (95% confidence interval, CI, 0.23 to 0.78) at 6 mo, 0.54 (95% CI 0.38 to 0.78) at 12 mo, 0.59 (95% CI 0.44 to 0.79) at 18 mo, and 0.67 (95% CI 0.52 to 0.86) at 24 mo. After we adjusted for the effects of PCP prophylaxis, zidovudine alone significantly reduced mortality at 6, 12, and 18 mo (relative risks 0.45, 0.59, and 0.70, respectively), but not at 24 mo (relative risk 0.81). Among zidovudine users, those who also used PCP prophylaxis before the development of AIDS had significantly lower mortality at 18 and 24 mo than those who did not (relative risks 0.62 and 0.60, respectively). CONCLUSIONS: The results of this study support the hypothesis that in HIV-1 infection, early treatment with zidovudine and PCP prophylaxis improves survival in addition to slowing the progression to AIDS.

Transfusion-Induced Changes in the Breathing Pattern of Healthy Preterm Anemic Infants—P Sasidharan, R Heimler. Pediatr Pulmonol 1992;3:170.

In order to evaluate the effects of transfusion on breathing pattern in growing, otherwise healthy, anemic preterm infants, we studied 14 infants whose mean (SD) birthweight was 1,212 (294) g, gestational age 29.28 (2.23) weeks, and hematocrit 25.86 (2.24)%. At a mean postnatal age of 41.35 (19.45) days, 12-hour nocturnal (transthoracic impedance) pneumocardiograms were obtained 24 hours prior to and after transfusion with 10 mL/kg of packed red blood cells. Posttransfusion pneumocardiograms revealed significant reduction of periodic breathing, of apnea episodes with 11-15 second duration, of apnea density, and of heart rate. These findings indicate that transfusion with packed red blood cells significantly alters certain cardiorespiratory variables in anemic preterm infants.

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Inhaled Albuterol Powder for Pulmonary Function Testing—E van Lunteren, A Coreno. Chest 1992: 101:985.

To assess the role of albuterol powder in testing for reversibility of airflow obstruction during routine pulmonary function testing, spirometric data from subjects with baseline $FEV_1/FVC < 70\%$ and $FEV_1 < 80\%$ predicted who received inhaled albuterol powder ($n = 42$) were compared with those who received isoetharine aerosol via metered dose inhaler ($n = 49$). Prebronchodilator lung function was comparable for the albuterol and isoetharine groups. With albuterol powder, 14 (33%) of 42 subjects showed reversibility of airflow obstruction (defined as a 15% or greater improvement in either FEV_1 or FVC) as compared with 30 (61%) of 49 subjects with isoetharine aerosol. The significantly ($p < 0.01$) lower rate of improvement with albuterol powder was especially prominent in subjects with moderate airflow obstruction (FEV_1/FVC of 45% to 59%). These data do not support the substitution of inhaled albuterol powder for isoetharine aerosol in assessing for reversibility of airflow obstruction during routine pulmonary function testing.

Evaluation of Accuracy and Reproducibility of Peak Flowmeters at 1,400 m—RM Gardner, RO Crapo, BR Jackson, RL Jensen. Chest 1992;101:948.

Peak flowmeters provide physicians and patients with objective measures about changes in pulmonary obstruction. We evaluated eight models of peak flowmeters and measured their accuracy and reproducibility with methods recently recommended by the National Asthma Education Program (NAEP). Waveforms from the American Thoracic Society's spirometer testing set were used to drive a computer-controlled syringe. Testing was done at Salt Lake City at an altitude 1,400 m. It appears that the original Wright peak flowmeter has been used as the "de facto" standard. We found that the original Wright peak flowmeter overestimated flows in its midrange; and, as a consequence, most of the other peak flowmeters also overestimated peak flows. The overestimation of peak flows may have been understated because of the 1,400-m altitude testing site. To the credit of the instrument manufacturers, we were pleasantly surprised with the quality, accuracy, and reproducibility of presently available peak flowmeters; however, as a result of our testing, we suspect that with little effort, manufacturers of peak flowmeters could improve the accuracy of their devices. Standardized testing methods and equipment should make the task of peak flowmeter design, manufacture, and testing even easier. We trust that manufacturers of peak flowmeters will respond appropriately and improve their instruments.

The Adult Respiratory Distress Syndrome: A Report of Survival and Modifying Factors—MR Suchyta, TP Clemmer, CG Elliott, JF Orme, LK Weaver. Chest 1992;101: 1074.

The adult respiratory distress syndrome (ARDS) is a form of acute lung injury characterized by arterial hypoxemia, reduced thoracic compliance, normal pulmonary capillary wedge pressure, and diffuse infiltrates on chest roentgenograms. Mortality remains high and has been associated with sepsis, organ failure, age and predisposing factors. We prospectively identified 215 ARDS patients over 34 mo to examine how these factors influence outcome. 102 (47%) of 215 patients survived. Age 65 years or older was associated with a survival of 34%, which was statistically different from the 53% survival of those patients younger than 65 years ($p = 0.02$). Aspiration pneumonia as a predisposing factor of ARDS was associated with a better survival ($p = 0.04$). Survivors had statistically less organ failure and sepsis than did nonsurvivors ($p < 0.05$). Cause of death was determined using the criteria of Montgomery et al for irreversible organ dysfunction. 45 (40%) of our patients died of respiratory failure (not sepsis). We conclude the following: (1) survival in our ARDS patients is different from previous reports; (2) the cause of death in our ARDS patients is different from that reported by Montgomery et al in 1985; and (3) multisystem organ failure, sepsis, age, and some predisposing factors of ARDS continue to be associated with decreased survival of ARDS patients.

Oxygen May Lower the O_2 Cost of Ventilation in Chronic Obstructive Lung Disease—ET Mannix, F Manfredi, P Palange, IRG Dowdeswell, MO Farber. Chest 1992;101:910.

It is known that the O_2 cost of ventilation (COV) in chronic obstructive lung disease (COLD) is high; O_2 administration to these patients lowers airway resistance, a major determinant of the COV. Thus, O_2 should lower the COV. We measured the COV in 10 stable COLD patients and 5 normal control subjects breathing room air and 30% O_2. Results indicate that the COV of our patients was elevated above that of control subjects, was related to disease severity, and was decreased with 30% O_2. The COV of control subjects also was lowered by O_2. At rest, O_2 lowered minute ventilation ($\dot{V}_E$), ventilatory equivalent for O_2 (VEQ O_2) VEQ O_2, and heart rate (HR). During submaximal exercise, O_2 lowered $\dot{V}_E$, reduced VEQ O_2, and

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extended total exercise time. An inverse correlation was noted between COV and maximal O_2 uptake. Thus, in stable COLD, the COV is elevated in proportion to the degree of airway obstruction, inversely related to exercise capacity, and lowered by O_2 administration.

Pulmonary Sequelae at Six Months following Extracorporeal Membrane Oxygenation—M Garg, SI Kurzner, DB Bautista, CD Lew, AD Ramos, ACG Platzker, et al. Chest 1992;101:1086.

Mechanical assisted ventilation for neonatal respiratory failure is associated with residual lung disease. Because ECMO *rests* the lungs, it has been suggested that ECMO will prevent chronic lung disease in survivors. To determine whether or not ECMO survivors have evidence of pulmonary sequelae, we studied 19 infants who were treated with ECMO for neonatal respiratory failure. Ten infants still required supplemental oxygen or pulmonary medications or both to treat clinical lung disease during the first 6 mo of life. Thoracic gas volume was normal. Pulmonary mechanics in ECMO survivors were compared with those of 13 preterm infants with BPD at similar age. We conclude that a significant proportion of ECMO survivors have residual abnormalities in pulmonary mechanics at 6 mo of age. We speculate that neonatal lung injury due to meconium aspiration and other causes is a more important determinant of abnormal pulmonary sequelae than the method of treatment.

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Estimation of Arterial Carbon Dioxide by End-Tidal and Transcutaneous P_{CO_2} Measurements in Ventilated Children—Y Sivan, MK Eldadah, T-E Cheah, CJL Newth. Pediatr Pulmonol 1992;12:153.

Simultaneous measurements of arterial, end-tidal, and transcutaneous carbon dioxide (P_{aCO_2}, P_{etCO_2}, P_{tcCO_2}, respectively) were obtained in 134 children receiving mechanical ventilation (ages 2 days to 16 years; mean 2.5 years). The mean (SD) P_{etCO_2} bias ($P_{aCO_2} - P_{etCO_2}$) was 3.4 (6.6) torr. When the P_{etCO_2} bias was plotted against the P_{aO_2}/P_{AO_2} ratio, a change in the scatter was obvious at about 0.3. The P_{etCO_2} bias for patients with P_{aO_2}/P_{AO_2} under 0.3 was 7.8 (7.3) torr compared to 0 (3.4) in patients with P_{aO_2}/P_{AO_2} above 0.3 ($p < 0.001$). P_{etCO_2} differed significantly from P_{aCO_2} ($p < 0.001$) only for patients with P_{aO_2}/P_{AO_2} under 0.3. The slope (P_{aCO_2} versus P_{etCO_2}) for these patients was 1.59, while the slope for patients with P_{aO_2}/P_{AO_2} above 0.3 coincided with the line of identity (1.00). The mean (SD) P_{tcCO_2} bias ($P_{aCO_2} - P_{tcCO_2}$) was -1.3 ± 7.2 torr. Skin perfusion was recorded at the area close to the transcutaneous CO_2 monitor electrode and was defined as normal when capillary refill was below 3 seconds. The P_{tcCO_2} bias for patients with normal skin perfusion was −0.2 (5.4) torr ($p = 0.73$) compared to −4.1 (9.9) for patients with decreased skin perfusion ($p = 0.01$). The slope of P_{tcCO_2} against P_{aCO_2} was closer to identity in patients with normal skin perfusion (1.17) than in patients where it was decreased (slope 1.40). We suggest that P_{aCO_2} estimation by both P_{etCO_2} and P_{tcCO_2} is sufficiently precise and reliable for clinical use in critically ill children.

Certain limitations stem from the nature of the techniques. Measurement of alveolar to arterial O_2 ratio may improve the precision of P_{aCO_2} estimation by capnography; assessment of skin perfusion is important in order to increase the accuracy of the transcutaneous method, especially in critically ill children.

Correcting for the Bernoulli Effect in Lateral Pressure Measurements—JHT Bates, PD Sly, J Sato, BLK Davey, B Suki. Pediatr Pulmonol 1992;12:251.

Measurement of airway pressure is essential in the study of respiratory mechanics, and is usually done via a lateral tap in the conduit (eg, endotracheal tube, cannula, or mouthpiece) leading into the subject's airway. Such pressure measurements, however, may be severely affected by the Bernoulli effect if the diameter of the conduit is small and the gas flow through it sufficiently high. We present in this note a simple method of assessing whether or not the Bernoulli effect is important in any particular situation. The technique involves comparing the pressure-flow relationships of the conduit obtained both by blowing air through it from one end and sucking air in the reverse direction by applying negative pressure at the same end. If the resistance of the conduit is the same for gas flow in both directions, then half the magnitude of the difference between the two pressure-flow relationships gives the magnitude of the Bernoulli effect pressure. We give results of an experimental situation in which this was the case. We also show that those conditions under which the Bernoulli effect is likely to be a problem are also those under which the velocity profile is likely to be approximately flat, thereby permitting the magnitude of the Bernoulli effect to be easily calculated.

Allergic Alveolitis in a 12-Year-Old Boy: Treatment with Budesonide Nebulizing Solution—K-H Carlsen, J Leegaard, OD Lund, H Skjærvik. Pediatr Pulmonol 1992;12:257.

Allergic alveolitis due to bird antigens was diagnosed in a 12-year-old boy. He suffered from cough, dyspnea, easy fatigue, anorexia, and

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severe weight loss. The diagnosis was verified by a gradual improvement when he was removed from the birds, exacerbation upon re-exposure, and the demonstration of serum precipitating antibodies against bird antigens. The patient recovered completely after a short course of oral prednisolone, treatment with inhaled nebulized budesonide for 3 months, and removal of the birds from his home.

Variability of Respiratory System Compliance in Mechanically Ventilated Infants—K Muramatsu, K Yukitake, T Oda. Pediatr Pulmonol 1992;12:140.

We measured respiratory flow, volume by integrated flow, and airway pressure in 4 mechanically ventilated infants. Compliance of the respiratory system (C_{rs}) was measured by the multiple occlusion method. Airway occlusion and release were achieved by a balloon in an occlusion valve between the endotracheal tube and a pneumotachometer, all connected in series with the respirator circuit. Values of C_{rs} varied greatly due to changes in mean airway pressure (MAP). The C_{rs} increased with the elevation of MAP on some occasions and decreased on others. Values of C_{rs} also varied corresponding to different occlusion pressures for individual levels of MAP. Thus C_{rs} changed continuously, even within a single respiratory cycle, the observed variability in C_{rs} was explained by the following mechanism: a pressure-volume (P-V) loop of tidal ventilation moves its position within the pressure-volume diagram and changes shape due to variations of the ventilator settings. In some infants the lungs were ventilated in the range of the linear portion of the P-V diagram while in others they may have been ventilated on the flattened portion. Some patients had convex expiratory P-V curves even with low pressure swings during tidal ventilation.

Nonlinear Pressure-Volume Relationship and Measurements of Lung Mechanics in Infants—GS Neto, T Gerhardt, A Silberberg, T Gerhardt, N Claure, S Duara, E Bancalari. Pediatr Pulmonol 1992;12:146.

We examined the effects of within-breath changes in compliance (C) upon the accuracy of measurements of compliance and resistance (R) by linear regression analysis and by Mead and Wittenberger's method. These effects were illustrated by a computer model and by lung models with linear and nonlinear pressure/volume relationships, and were also studied in 14 normal spontaneously breathing premature infants (mean [SD] birthweight 1,290 [200] g, gestational age 29.9 [2.7] weeks, age 7.4 [2.1] days). Flow was measured by pneumotachography and tidal volume was derived as digitally integrated flow, and transpulmonary pressure as airway minus esophageal pressure. We found that C and R calculated from the equation of motion is accurate only if C and R remain constant throughout the respiratory cycle. Calculated compliance depends more on C at the end than at the beginning of inspiration. A decreasing C leads to underestimation of R, while an increasing C leads to an overestimation of inspiratory R. Calculated total R may be accurate, but with low r values for measurement points. Mead and Wittenberger's method and the regression method are similarly affected by changing C; however, since the regression method is based on many more measurement points and therefore allows the detection and analysis of within-breath changes of C and R, it is less prone to erroneous results secondary to signal artifacts than Mead and Wittenberger's method.

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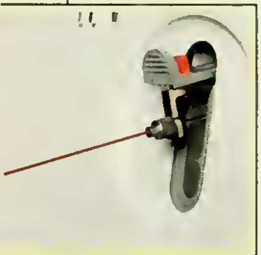

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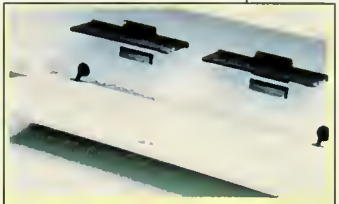

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ACLS Skills for the Respiratory Therapist: Time for a Mandate

Respiratory therapists work throughout the hospital—in the emergency department, critical care units, recovery room, and general wards. They respond to all resuscitation calls and often participate in the unannounced 'codes' that occur in critical care units. Therapists respond to calls from the emergency department to assist with advanced life support in trauma cases and to treat the acute respiratory emergencies that often contribute to cardiac arrest. And, yet, we find from our own experience and the experience of others[1] that Advanced Cardiac Life Support (ACLS) training and certification are not universally accessible to or achieved by therapists. This is in spite of the fact that almost 1 million health care providers were certified by the American Heart Association from July 1986 to July 1991 (personal communication, American Heart Association National Center, Dallas TX, April 1992). We believe that the involvement of respiratory therapists in emergency and critical care mandates ACLS training and certification.

The Therapist as a Primary Member of the Resuscitation Team

Respiratory therapists are primary members of hospital resuscitation teams because of their special training and education in airway control, ventilation, and administration of supplemental oxygen. Specific knowledge and skills include bag-valve-mask ventilation, tracheal intubation, drug administration via tracheal instillation, use of transport ventilators, post-resuscitation mechanical ventilation, hemodynamic monitoring, and use of capnography. The therapist obtains and interprets arterial blood gas values and fully appreciates the oxygen transport problems that occur when basic life support is interrupted for advanced life support interventions.

Utilizing the Therapist's Training and Capabilities

It has been shown that during ACLS, paramedics intubate the trachea with a 90% success rate,[2-5] whereas the skill can be quickly lost if not practiced.[6] With their basic training reinforced by ACLS training and the opportunities presented to maintain proficiency, respiratory therapists should be able to intubate with a success rate similar to that seen in paramedics.

Many persons in nontraumatic cardiac arrest experience ventricular fibrillation within a few minutes after collapsing.[7] The chances of the post-shock rhythm's being a sustained, perfusing one are increased by early delivery of the defibrillation shock.[8] To achieve early defibrillation, the American Heart Association recommends that all responding emergency personnel (both hospital- and community-based) be trained to recognize ventricular fibrillation and to defibrillate when the victim's condition warrants.[9] We know (from our own experience and from communication with instructors over the years) that recognition and treatment of ventricular fibrillation and pulseless ventricular tachycardia are standard components of respiratory therapy curricula and that laboratory practice with defibrillators is provided in many programs. Thus, participation in early defibrillation is a logical extension and utilization of the therapist's training.

Where Is the Greatest Need?

It may not be practicable to have every respiratory therapist ACLS trained and certified. However, ACLS training makes sense for those therapists who frequently assist with resuscitation—those who respond to code calls, cover the emergency room, work in critical care units, and serve on transport teams.

Some of the need for training and certification may be institution specific. Large teaching hospitals are likely to have an anesthesiologist on the resuscitation team. However, the therapist still has an important role in continuing airway management when the anesthesiologist returns to the operating room. In some large hospitals, the therapist assumes other ACLS responsibilities—providing chest compression, connecting cardiac monitor leads, and preparing and using the defibrillator. Although the number of cardiac arrests occurring in community and rural hospitals may be smaller, the therapist often assumes more responsibility on the resuscitation team—particularly during the night and on weekends when the operating room is inactive and no anesthesiologist is available to respond. The respiratory therapist and nurse on a ground- or air-transport team may be faced with a patient in arrest and the need to provide all facets of ACLS.

Of course, additional specialized needs exist. In addition to participation in ACLS, pediatric and neonatal practitioners should participate in the AHA's Pediatric Advanced Life Support, or PALS, program and the Neonatal Resuscitation Program, or NRP, now administered by the Academy of Pediatrics; certain critical care therapists may need to participate in the Advanced Trauma Life Support, or ATLS, program of the American College of Surgeons designed for physicians who do not routinely care for the injured.

Accomplishing the Tasks of Certification and Recertification

Health care professionals may tend to overestimate their resuscitation skills and knowledge, and skills tested 6 and 12 months after training have been shown to deteriorate to near pretraining levels.[10] Yet, a survey of 60 physicians, nurses, and paramedics who had completed ACLS courses within the previous 2 years found that 90% believe that the ACLS training helped them provide better patient care.[11] The respiratory therapist as a primary member of the resuscitation team should be ACLS trained and certified and have ready access to the necessary retraining.

What Does It Cost To Certify and Recertify?

The cost of ACLS courses varies from $125-$200 for each person trained and certified. A department with a staff of 50 seeking ACLS training for all would be faced with an annual cost of about $5,000 (training or retraining for one half of the staff each year). Less expensive yet effective routes to recertification include courses that emphasize remediation for problem areas identified in pretesting (eg, assessment, defibrillation, or drug therapy)[12] and modular self-paced, problem-oriented learning packages and computer-assisted instruction.[13-15]

The Role of the Medical Director

The medical director of the respiratory care department should help identify staff members who need ACLS training and support the technical director and supervisors in justifying the need for ACLS training. The medical director should actively participate in ACLS training and evaluation. And, finally, we believe that medical director should be a role model by maintaining his or her ACLS certification and if practicable serving as an ACLS faculty member.

The Role of the Technical Director and Supervisors

The technical director and supervisors should support maintenance of ACLS skills and knowledge by organizing resources for continuing education and time for training. Modular units for independent study with manuals, flashcards, workbooks, videotapes, mannequins, and audiotape-slide packages should be readily available to staff members. Competency evaluation of ACLS knowledge and skills should be part of the department's quality assurance program, and the director and supervisors should serve as role models by maintaining their own certification.

The Role of Respiratory Therapy Programs

The faculty of each respiratory therapy school should make certain its curriculum provides the content necessary for students to gain the knowledge and skills that they will need to successfully complete an ACLS course. Common mistakes made by faculty members who teach cardiopulmonary resuscitation skills include failing to teach in a standardized way, limiting practice time, failing to correct errors in performance, and using checklists inappropriately.[16] Because ACLS courses are usually offered intensively over 2 consecutive days, the faculty should prepare students by reviewing the units of study for several weeks before the course begins. Students should be scheduled for ACLS instruction after they have completed Basic Life Support certification, have finished their critical care rotation, and have observed several resuscitations. Thus, students should be assigned to an ACLS course shortly before graduation in order to take full advantage of the program courses that provide the knowledge base for ACLS. It is important for respiratory therapy programs to provide this instruction because it has been shown that health care providers with ACLS training perform more competently, have more confidence, and exhibit less anxiety during resuscitations.[17,18] Further, the graduate with ACLS certification is more 'marketable.'

Importance of ACLS Training to the Profession and to the Patient

Advanced cardiac life support is a team effort and demands a high level of interprofessional communication. Physicians, nurses, and respiratory therapists must understand the role of each team member. The team effort needs to come together in less than 4-8 minutes to improve the patient's chances for long-term survival.[19] ACLS courses remain interdisciplinary and standardized to promote a common understanding of the algorithms used to manage cardiac arrest and life-threatening arrhythmias. All ACLS candidates have the same pass/fail criteria applied to their performance regardless of their professional role or job title. This leads to common ground and mutual respect among team members.

Respiratory therapists have knowledge and skill indispensable to ACLS teams. However, to remain effective the therapist must seek and maintain ACLS certification. This is a credential that other team members immediately respect because it implies knowledge of overall management. This respect and increased professional recognition lead to increased ACLS responsibility and job satisfaction.

Respiratory care professionals have always placed the patient first and, consequently, are constantly looking for ways to work more closely with physicians and nurses to improve patient care. ACLS training and certification for the respiratory therapist is one more way to achieve that goal. It's time to prepare for more responsibility on the resuscitation team—for the patient's sake!

Thomas A Barnes EdD RRT
Director of Clinical Education
for Respiratory Therapy
Director of Cardiovascular
Technology Program
Associate Professor
Northeastern University
Boston, Massachusetts

Charles G Durbin Jr MD
Professor of Surgery
and Anesthesia
Medical Director of
Respiratory Therapy
University of Virginia
Medical Center
Charlottesville, Virginia

Reprints: Dr Thomas Barnes, Dept of Cardiopulmonary Sciences, Northeastern University, 360 Huntington Ave, Boston MA 02115.

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EMERGENCY RESPIRATORY CARE
Part I

A Special Issue

based on a conference
held October 3-5, 1991,
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Dear Reader:

The American Association for Respiratory Care and its science journal Respiratory Care are proud to present the June and July 1992 issues containing the proceedings from the 1991 Journal Conference. The Conference dealt with topic important to all of us—emergency respiratory care. The papers and discussions presented here are well-organized, state-of-the-art materials designed for you to use in your daily practice.

Considerable planning and work are required to present and publish the proceedings of a conference like this. We are grateful to the speakers, the Editorial Board, Conference Co-Chairmen Thomas A Barnes EdD RRT and Charles G Durbin Jr MD, Editor Pat Brougher, and Editorial Coordinator Donna Stephens for planning, writing, editing, and participating in the Conference and sharing this important information with us.

We are also indebted to Allen & Hanburys, Division of Glaxo Inc, for their generous grant to the Association, which made possible the presentation and publication of the Conference proceedings. Allen & Hanburys has supported the last four Journal Conferences and for this we will always be thankful.

Sincerely,

Ray Masferrer

Ray Masferrer RRT
Managing Editor
Respiratory Care

The Role of the Respiratory Therapist in Emergency Care

Robert M Kacmarek PhD RRT

Introduction

A universal definition for the role of the respiratory therapist in any specific aspect of medical care is difficult to formulate. Many factors, both within and external to each respiratory care department, contribute to this role definition. Emergency care is diverse, dynamic, and demanding, and presents opportunities to which respiratory therapists can contribute significantly. Provision of emergency care within the hospital is a team effort. The respiratory therapist, the physician, and the nurse comprise the three primary components of this team. To fully contribute, the therapist must be able to communicate at the level expected by the medical staff and be willing to aggressively provide consultation in those situations in which his or her knowledge of respiratory care exceeds that of other health care workers attending a given patient. The therapist is the consultant on both traditional and newly developed respiratory care techniques, and the consultant role is universal, regardless of the institution.

Establishing the need for an expanded role for the respiratory therapist that encompasses technical tasks beyond those normally performed is more difficult. It is in technical applications that diversity of roles exists. In order for role expansion to occur in a given institution, the respiratory care department must first identify the need for expansion; second, establish the ability of the respiratory therapist to safely perform the task; and, third, ensure that the change in role definition is cost-effective. For example, it may be difficult to justify training respiratory therapists to perform emergency endotracheal intubations in large university institutions where anesthesia residents outnumber staff therapists; however, justifying the need for respiratory therapists to perform emergency intubations in small community hospitals is relatively easy.

Emergency care encompasses most aspects of what we can call traditional respiratory care (Table 1). In this paper, I define the role of the therapist in each of these aspects and attempt to show where the role of the therapist may differ in university versus community versus rural hospital settings. Finally, I discuss issues regarding the staffing necessary to provide the described care and the need to establish the role of the respiratory therapist by documenting therapist efficacy through research. In preparing this paper, I was unable to find a single article that documented the capabilities of the respiratory therapist to provide any aspect of emergency care.

Patient Assessment

As outlined by MacIntyre,[1] an often neglected but essential aspect of all patient care is physical

Table 1. Aspects of Emergency Care in which Respiratory Therapists Play a Major Role

Patient assessment
Oxygen therapy
Drug delivery via the respiratory tract
Airway care
Mechanical ventilation
Cardiopulmonary resuscitation
Transport

Dr Kacmarek is Director, Respiratory Care, Massachusetts General Hospital, and Assistant Professor, Department of Anesthesiology, Harvard Medical School—Boston, Massachusetts.

A version of this paper was presented by Dr Kacmarek on October 3, 1991, during the RESPIRATORY CARE Journal Conference on Emergency Respiratory Care held in Cancun, Mexico.

Reprints: Robert M Kacmarek PhD RRT, Respiratory Care, Ellison 4, Massachusetts General Hospital, 14 Fruit St, Boston MA 02114.

assessment. In the emergency setting, the respiratory therapist's ability to assist in assessment of the upper and lower respiratory tract is critical (Table 2). Patients frequently present with asthma or foreign-body obstruction that requires careful assessment of their respiratory status. In addition, the respiratory therapist's expertise is useful in clinically differentiating between croup and epiglottitis in children, especially in settings in which physicians are not immediately available (eg, rural and community hospitals). Finally, regardless of the specific presenting condition, evaluation of ventilatory pattern, accessory muscle use, airway patency, and work of breathing by a health care worker whose focus is on the cardiopulmonary system (ie, the therapist) is invaluable.

Table 2. Patient Characteristics To Be Assessed in Emergency Care

Ventilatory pattern
Use of accessory muscles
Airway patency
Work of breathing
Peak inspiratory and expiratory flows
Timed, forced expired volume
Maximum inspiratory pressure
Vital capacity
Flow-volume loops
Airway pressure, flow, and volume waveforms
Pressure-volume loops

Quantitative assessment of pulmonary function and ventilatory mechanics is becoming commonplace in the emergency ward. Determination of peak inspiratory and expiratory flows and FEV_1 in patients presenting with acute asthmatic attacks assists in quantifying not only the severity of the exacerbation but also the efficacy of treatment. Evaluation of ventilatory reserve by making maximum inspiratory pressure or vital capacity measurements assists in quantifying the extent of pulmonary, neurologic, or neuromuscular dysfunction. Newer instruments (such as the Bicore CP-100,[2] Fig. 1, and the Med-Science VenTrak,[3] Fig. 2), although designed for intubated patients, do allow for detailed immediate bedside assessment of ventilatory function. Such devices not only allow continuous monitoring of airway pressure, flow, and volume waveforms but also can generate pressure-volume and flow-volume loops. These devices are easy to operate and can provide data within minutes. As these and other instruments become more available, it is not unreasonable to predict their common use by respiratory therapists in the emergency ward, as well as in intensive care units.

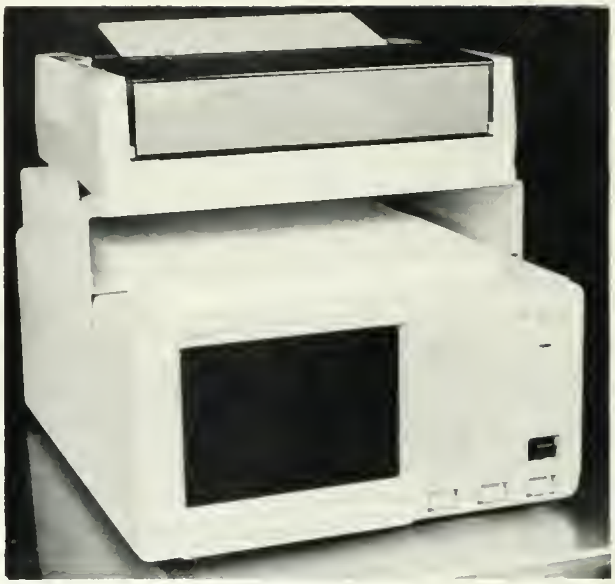

Fig. 1. Bicore CP-100 Pulmonary Monitor.

Fig. 2. Med-Science VenTrak respiratory monitor.

Oxygen Therapy

Historically, the cornerstone of respiratory care has been oxygen therapy. Although oxygen is one of the most common drugs prescribed in the hospital setting, it is also one of the most poorly controlled drugs administered.[4] Need for precise, accurate delivery of high concentrations of oxygen is common in the emergency setting. Not infrequently, practitioners presume that a given oxygen delivery system provides consistently higher F_{IO_2} in the face of marked ventilatory demands than is likely. The proper use of high-flow oxygen-delivery systems not only may assure better patient oxygenation but also can avoid more invasive treatment to correct hypoxemia. It is unrealistic to assume that anyone other than the respiratory care practitioner has detailed knowledge of the capabilities and the variability of high-flow systems.[5,6] It is essential for the therapist to function as a consultant in the prescription and administration of oxygen therapy.

The respiratory therapist is also invaluable in outlining and defining the capabilities of home oxygen-delivery systems. Particularly in rural or community hospital settings, the respiratory therapist may be the only practitioner with knowledge of the idiosyncrasies of demand oxygen-delivery systems, reservoir nasal cannulas, or transtracheal oxygen therapy. Presentation of patients with these appliances can present management difficulties unless knowledgeable respiratory therapists are available for consultation.

Drug Delivery via the Respiratory Tract

Traditionally, aerosolized sympathomimetics, anticholinergics, mucolytics, and even steroids have been delivered by respiratory therapists in the emergency ward. More recently the respiratory route for drug delivery has been explored in the setting of cardiopulmonary resuscitation (CPR). Particularly when peripheral or central venous access to the circulatory system is unavailable, atropine,[7] lidocaine,[8] epinephrine,[9] and naloxone[7] have been instilled into the endotracheal tube during CPR. Although results are variable, data imply that instillation may be an effective alternate route of administration during CPR. Drug delivery via this route is easily performed by the respiratory therapist during manual ventilation.

It has been well documented that the effective dose of an aerosolized drug can vary, especially in the presence of an endotracheal tube.[10] However, recent research implies that enhanced drug delivery by metered dose inhaler (MDI) can be obtained by administering the MDI via an extension nozzle at the tip of the endotracheal tube. Peterfreund[11] and Taylor and Lerman[12] have reported and our unpublished data confirm that up to 90% of the MDI dose is delivered to the lower respiratory tract when the MDI is actuated through a 19-gauge 12-inch catheter at the tip of the endotracheal tube. This delivery methodology appears particularly promising in the acute care and emergency settings. Again, the respiratory therapist is the ideal clinician to deliver drug therapy by this route.

Mechanical Ventilation

The respiratory therapist is a vital component of the emergency medical team during the initiation of mechanical ventilatory support. Therapists must ensure proper ventilator function, set the desired ventilatory pattern during (and subsequent to) initiation, and modify ventilator settings based on patient response. The role of the respiratory therapist is undisputed in this aspect of emergency care.

Recent data[13-15] suggest that the respiratory therapist will be applying noninvasive positive pressure ventilation in the emergency room on a regular basis. The initial successful use of noninvasive ventilatory techniques to reverse hypercapnia and prevent endotracheal intubation in the emergency setting is very promising. Brochard et al[13] compared the application of positive pressure ventilation via face mask in 13 patients with acute exacerbations of chronic obstructive pulmonary disease (COPD) to 13 matched controls who did not receive ventilatory support. Only 1 of 13 in the treated group required endotracheal intubation; however, 11 of 13 in the control group required intubation. In addition, as compared to the control group, the treated patients had a more transient need for ventilatory assistance [mean (SD), 3 (1) vs 12 (11) days, $p < 0.01$] and a shorter stay in the intensive care unit [7 (3) vs 19 (13) days, $p < 0.01$]. Brochard et al[13] used a 'home-made' positive-pressure generator to apply a pressure and flow waveform similar to pressure support by full face mask. Similar but uncontrolled data have also been reported by Meduri et al[14] who were able to avoid intubation by applying ventilation via face mask with a Puritan-Bennett 7200 ventilator. All patients in the series were initially maintained on pressure support (10-20 cm H_2O) and continuous positive airway pressure (CPAP) to achieve a ventilatory rate < 25/min and a tidal volume (V_T) > 7 mL/kg. Patients unable to tolerate pressure support received synchronized intermittent mandatory ventilation (SIMV). More recently, Marino[15] reported on the use of a nasal CPAP mask and a Bear 2 ventilator for the provision of noninvasive mechanical ventilation during acute ventilatory failure; 9 of 13 patients with COPD or hypothyroidism responded to treatment.

The introduction of the Respironics BiPAP[16] positive pressure ventilator (Fig. 3) and its reported successful application in acute[17] as well as chronic ventilatory failure[18,19] makes the application of noninvasive positive pressure ventilation in the emergency setting even more feasible. Although this unit is not ideal (ie, no alarms, attachments required for pressure monitoring and V_T measurement, and oxygen concentration difficult to titrate), it is generally quickly accepted by patients. Its continuous flow and ability to compensate for leaks seem to make it ideal for noninvasive nasal ventilation.

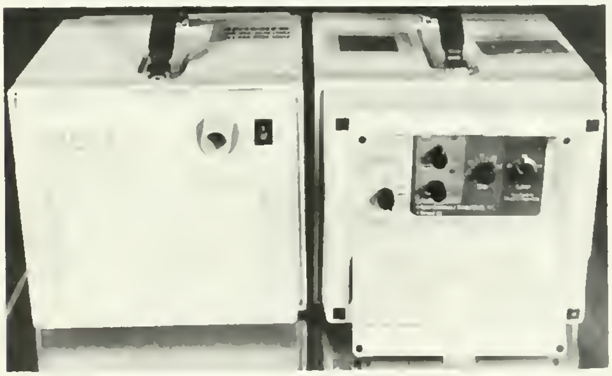

Fig. 3. Respironics BiPAP positive pressure ventilator.

For the successful noninvasive application of positive pressure ventilation, respiratory therapists must be specially trained in its technical and clinical application. For example, therapists must spend long periods of time one-on-one with the patient to ensure patient acclimation to and proper adjustment of the device and must properly size the mask to help assure success. The use of noninvasive mechanical ventilation techniques in patients who present to the emergency room with acute ventilatory failure superimposed on chronic ventilatory dysfunction, presents a therapeutic opportunity in which the respiratory therapist can play an important role.

Airway Care

Maintenance of endotracheal or tracheostomy tubes in emergency (as well as critical care settings) has long been part of the role of the respiratory therapist. Evaluations of tube placement, tube manipulation, and measurement of cuff pressure and volume are routine functions carried out by respiratory therapists.[20] The extent to which endotracheal intubation in the emergency setting is performed by respiratory therapists varies from institution to institution. However, it is my observation that almost all registry-level respiratory therapy programs instruct students in the technique.[4,21,22] The argument for intubation by respiratory therapists can be carried out on several levels: (1) Do respiratory therapists have the educational background to perform this technique? (2) Is there a need for therapists to be trained in intubation? (3) Is it safe for nonphysicians to perform intubation? As already noted, training in intubation techniques is part of the curriculum of most registry-level respiratory therapy programs. This, coupled with extensive coursework in pulmonary anatomy and physiology, seems to answer the first question positively. In fact, respiratory therapists probably receive more contact hours of coursework on this subject than any other nonphysician health care worker. The question of whether there is a need for respiratory care practitioners to perform endotracheal intubation is more difficult to answer. Clearly, in large university settings where resident staff on all shifts outnumber respiratory care staff, the need for respiratory therapists to perform endotracheal intubation does not exist. However, in rural and community hospital settings the respiratory therapist is frequently the practitioner most qualified to provide respiratory and airway care. In these settings, it is clear that a need does exist and is being filled by respiratory therapists. The actual performance of endotracheal intubation by respiratory therapists extends beyond the rural and community setting to both adult and neonatal transport teams and to many Level-III nurseries. Finally, is it safe for respiratory therapists to perform endotracheal intubation? Unfortunately, no available data evaluate the effectiveness of respiratory therapists in intubation. However, data on performance of intubation in emergency settings by paramedics suggest that the technique can be safely performed by nonphysicians.[23-27]

The first report of endotracheal intubation efficacy by paramedics was presented by DeLeo[24] in 1977. Table 3 summarizes these results compared to those of physicians (Heartmobile) in the field. As noted, they compared equivalently, with about 90% of attempted intubations successful. Jacobs et al[27] reported a 96.6% success rate by paramedics, with only 6 failures out of 172 intubations, while Stewart et al[25] reported successful intubations in 701/779 patients (90%). The frequency of complications in these studies was also limited. DeLeo reported 40 incidents of vomiting and potential aspiration, whereas Jacobs et al reported only 3 incidents of vomiting. Table 4 lists the more detailed complication rate reported by Stewart et al.[25] In this series, 3 patients presented to the emergency ward with esophageal intubations; however, review of each case revealed a break in protocol. Tube placement was not verified by auscultation of the chest and abdomen in any of the 3 patients. O'Brien et al[23] have recently reported a 71% success rate in blind nasotracheal intubation by paramedics in a series of 324 spontaneously breathing patients. Table 5 lists the complication rate in the O'Brien study; again, only three major complications were noted. In all these series, esophageal intubation was the common major complication listed. However, none of these groups indicated that end-tidal CO_2 detection devices (eg, Fenem FEF, now EasyCap, Nellcor, Hayward CA) were used. Recent data indicate that the frequency of unrecognized esophageal intubation was greatly reduced with these devices,[28,29] although their performance during CPR is still questionable.

Table 3. Successful Orotracheal Intubation by Paramedical Personnel vs Physicians*

Year	Attempted	Successful	% Successful
Paramedics			
1972	199	169	84.9
1973	266	245	92.1
1974	320	297	92.8
1972-1974	785	711	90.6
Physicians			
Heartmobile	44	39	88.6

*Reprinted, with permission, from Reference 24.

Table 4. Orotracheal Intubation Complications Rate in Field Intubations by Paramedical Personnel*

Complication	Number Reported	% of Attempted Intubations
Prolonged attempt (> 45 s)	42	5.4
Esophageal placement (corrected)	11	1.4
Esophageal placement (not corrected)	3	0.4
Vomiting	7	0.9
Trauma soft tissue	3	0.4
Loose teeth	3	0.4
Right main-stem (not corrected)	3	0.4
Other	2	0.3
None	705	90.5

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Table 5. Complication Rate in Blind Nasotracheal Intubation by Paramedical Personnel*

Complication	Number Reported	% of Attempted Intubations
Minor		
Epistaxis	25	7.7
Cuff leak	6	1.9
Need to reposition	5	1.5
Sinusitis	3	0.9
Local necrosis	1	0.3
Pneumomediastinum	1	0.3
Major		
Pyriform sinus tear	1	0.3
Esophageal intubation	2	0.6

*Reprinted, with permission, from Reference 23.

The data summarizing the paramedic's experience clearly indicate that endotracheal intubation by nonphysicians can be performed safely with a high success rate. The success rate of paramedics in the field is less than the success rate of anesthesiologists in the operating room. However, if we consider the circumstances under which paramedics must function, a 90-95% success rate is exceptional. Based on these data, it is appropriate to assume that respiratory therapists, properly trained, will be able to function as well. This is especially true of emergency in-hospital intubation because the majority of such intubations are performed during resuscitation or on comatose patients. Few trauma patients would require intubation because most present to the emergency ward already intubated. Complication rates can be diminished from those listed by mandatory monitoring of exhaled CO_2 to verify intubation. CO_2 monitoring is not 100% accurate in CPR settings; however, combining CO_2 monitoring with proper post-intubation assessment should eliminate unrecognized esophageal intubations. As noted earlier, the need for universal practice of emergency intubation by respiratory therapists is difficult to justify because of the presence of anesthetists in large university hospitals. However, the need does exist for respiratory therapists to intubate in small community and rural hospitals, in Level-III nurseries, and during transport. Before endotracheal intubation becomes universal, even in these settings, it is essential that the experience be published from centers at which therapists intubate. At present, no data are available to demonstrate the efficacy of respiratory therapists' performing endotracheal intubation.

CPR and Defibrillation

Respiratory therapists are an integral part of all inpatient CPR teams. Establishment of an airway and provision of manual ventilation and, in some settings, endotracheal intubation are clearly defined roles for respiratory therapists. In addition, if more than one respiratory therapist is present at the arrest scene, cardiac compression is normally carried out by one of the therapists. Nurses and physicians are most frequently involved with evaluation of electrocardiac response and drug therapy. In those settings in which attendance at a cardiac arrest is predominantly staffed by nurses and therapists, it seems reasonable to expand the role of the therapist to include defibrillation. This would not only enhance the contribution of the therapist during CPR but would free nursing personnel to focus on evaluation of electrocardiographic patterns and drug therapy. Again, no data are available evaluating the ability of therapists to perform this task. However, defibrillation is a common task performed by paramedics,[30] and with appropriate training could easily be performed by respiratory therapists.

CPR and ACLS Training

The CPR training for RCPs is mandated by the JCAHO.[31] No such requirement exists for advanced cardiac life support (ACLS) training. However, as outlined, especially in rural and community hospital settings, the need for ACLS certification may be acute. It is difficult to argue against such enhancement of educational and technical capability in settings in which a limited number of physicians are available to perform ACLS. Respiratory therapists are an integral part of the CPR team, and as such require the same level of training as that received by the other two primary team members (nurse and physician). ACLS certification of all respiratory therapists is a monumental task, considering the cost and availability of courses; however, in order to increase the effectiveness of the respiratory therapist in emergency care, it is essential. I call for ACLS training to be an integral part of the curriculum in respiratory therapist programs. If every new graduate therapist were ACLS certified, the number of ACLS instructors and ACLS courses outside of the school would have to be expanded, allowing greater access for practicing therapists. ACLS certification is the single most critical factor in properly establishing the therapist role in emergency care.

Intrahospital Transport

The role of the respiratory therapist in the intrahospital transport of the acutely ill, recently stabilized emergency care patient is critical. Most transport-related problems are associated with the basic components of CPR, airway stabilization, breathing, and cardiovascular stability. Other than the anesthetist, the therapist is the individual most qualified to maintain the patient's airway and to perform ventilation. If a transport ventilator is being used, the therapist should be at the head of the bed to ensure the unit's function and to make airway manipulations where necessary. In addition, the therapist should be actively involved in the evaluation of the patient's cardiopulmonary status.

Staffing for Emergency Care

It is difficult to define staffing criteria for all institutions. Individualized staffing patterns must be established, based upon the defined institutional role of the therapist. In large institutions with active emergency wards, the assignment of a therapist exclusively to the emergency ward may be appropriate. In others, a light routine assignment along with the emergency ward assignment may be ideal. Whatever the circumstance, the distribution of manpower should be appropriately based on volume of care and the role of the therapist if that role is to be continued. We have found it better not to assign responsibility for first response to cardiac arrest elsewhere in the hospital to the emergency ward therapist to ensure that disruption of the role played by that therapist does not occur.

In Conclusion

The respiratory therapist's role in emergency medicine can be important but as yet is not universally defined. Respiratory therapists play a primary role in oxygen and aerosol therapy, evaluation of the cardiopulmonary system, airway care, and ventilatory management. There is also a role for the therapist in intubation and defibrillation, especially in rural and community hospitals, but respiratory care departments must be aggressive in assuming that role. Opportunities for an enhanced role for the respiratory therapist exist but require significant individual, as well as group, effort to achieve. In order to achieve these goals, all respiratory therapists must become ACLS-certified. ACLS certification should be part of the curriculum of all respiratory therapist programs. In addition, we must publish the results of our experiences in emergency care. Data are necessary, especially regarding the therapist's ability to intubate patients in the emergency setting. Unless we document and market our ability to function, it will be difficult for our role to develop properly.

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Kacmarek Discussion

Nemiroff: One aspect that you briefly touched on—a theme that I'll enlarge on later in the team role of the respiratory therapist—is the need for aggressiveness and for being an information resource for physicians practicing in the emergency department. The therapist's initial approach to the patient (and he or she may be the first one to see the emergency patient) sets the tone for the rest of the resuscitation, particularly in near-drowning. I'd like to amplify that and hope the therapist would take on the role and set the tone for resuscitation. To your knowledge, has anything been written about that?

Kacmarek: No, but I think you're correct. We've failed to function in the primary consulting role for which we are suited in many emergency situations. Therapists, for whatever reason, perhaps because of their position in the hierarchy of the medical community, are not always as aggressive as I think they should be in expressing their opinions on ways to manage patients in these settings. Frequently, because of, as you say, their narrow perspective in relationship to the wide perspective of the emergency medical physician, therapists may be able to consult and advise very specifically on better ways of handling a situation. It's something that we as respiratory care professionals need to work on.

Giordano: Bob, are you familiar with the pilot project that was concluded just a short time ago in New York City, regarding the role of respiratory care practitioners in administering I.V. drugs in the emergency room?

Kacmarek: No, I'm not, Sam. I don't know anything about it.

Giordano: It's my understanding from some individuals in the area that the pilot project was undertaken at the behest of HANY (which is the Hospital Association of New York) as a response to the severe nursing shortage in that area. With the state's blessing they went about 'flexing,' if you will, licensure requirements to allow persons in the hospital deemed skilled and trained to carry out these procedures in a pilot project to see if such individuals could play a greater role. In this particular instance, it was I.V. insertion and steroid administration to asthmatic patients (personal communication from ES Salsberg, Director, Bureau of Health Resources Development, Department of Health, State of New York to AP Mezey MD, Medical Director, Bronx Municipal Hospital Center, Bronx NY, Dec 31, 1991).

Kacmarek: No, just one quick comment. I'm not aware of any data on that. The only role that I know that therapists play (and it's sporadic) in administering pharmacologic agents other than via the aerosol route, is during ECMO. Our ECMO team is composed entirely of therapists, and therapists administer all drugs that enter the ECMO system.

Wilson: I really can't comment on the project that you cited. I do have a related question for Bob. With the metered dose inhalers (MDIs), you suggest that this may be a more efficient, more predictable way of delivering drug, and because it is obviously related to the mechanical activities that therapists are involved in, it seems a natural assumption that they should be directly involved in the use of this device. In an emergency setting, especially when dealing with asthmatics, being able to predict the amount of drug is not necessarily that important because if it doesn't work (whether it's intravenous, subcutaneous, or any other route) you assess and then either increase the delivery or add an additional drug. But, given your proposed expanded role for the therapist, how do you go about training a department of therapists to be as facile and knowledgeable as necessary to carry out that therapy appropriately?

Kacmarek: I would approach training them in the same way that I would approach training for any new aspect of respiratory care. Trying to get my whole staff competent in performing noninvasive nasal positive pressure ventilation has been a task, I must admit. It's not just learning the new technique, it's trying to get people to approach the patient in the appropriate manner psychologically because a lot of it is interaction with the patient. First of all, you set up inservices in which you present didactic information; you then evaluate each individual's ability to understand and retain that information through examination; but also you need to develop a methodology for determining whether the therapists have developed the technical capability, and that really has to be a one-on-one evaluation. You provide them a procedure, watch them perform, correct their behavior, and continue to have them demonstrate performance until you're assured that the technical performance is within the guidelines that you've outlined. That's the approach we've taken with aspects of respiratory care in which it is critical that the technique be precisely performed. I think it's important to remember that it is easier to take a group of respiratory therapists (50, or 60, or 70) in a given institution and train them to perform a technique that fits very nicely into the scope of their defined practice than it is to take 1,500 or 2,000 nurses and train them in a technique that is really on the periphery of or may be totally outside their practice. I don't mean to imply that getting practitioners to accept new techniques and to perform them properly is the easiest thing to do—although I think it's manageable, primarily because of the numbers and the close control that exists and the repetitive nature of their performing the task. We've been able to set up programs, to teach didactically, to have demonstrations in laboratory settings, and to evaluate in the clinical setting the ability to perform a given task.

Reines: Bob, I agree that there are absolutely no data available on intubation options for therapists. Because most of the programs are teaching intubation and because, in the university centers, this is frequently a cause of frustration, I wonder if the data don't need to be gathered and if the AARC isn't the body to do it? Barry Shapiro of the National Association of Medical Directors of Respiratory Care, in an informal poll of respiratory care practitioners, found several years ago that indeed in hospitals smaller than 200 beds a lot of the intubations were being performed by therapists, whereas in university centers they are not. I think we need some data because obviously the EMT data show that it can be done by nonphysicians. Similarly, there are life packs and smart defibrillators that respiratory care practitioners can easily manage in a code situation. I think the AARC needs to define some of these and set up some guidelines. Maybe the guidelines and standards committee could look at it.

Kacmarek: I agree 100%. We have failed in this area because we have no data to demonstrate that we have this skill, which we are in fact teaching students.

Hurst: One of the things we as physicians need to realize is that major interfaces are made a lot simpler by physician advocacy. When I first moved to Cincinnati, the respiratory therapists in the surgical intensive care unit were not allowed to draw samples for blood gas analysis from arterial lines. To this very day, unfortunately, the medical service does not enlist the assistance of respiratory therapists in the ICU. In this day and age it is unbelievable, but happens to be a fact. One of the things that we can do is to make sure that respiratory care practitioners are included as part of the health care team. In the ICUs this is critical. They actively participate in rounds in our institution. During the presentation of patients at daily rounds—both on the surgical service and in the pulmonary rehabilitation units—their input is very important in determining overall patient care plans. Therefore, part of that responsibility is ours.

Kacmarek: I agree—so does our staff— but I think a lot of respiratory therapy departments, unfortunately, have not pushed the issue with the medical staff or the medical director of their departments to allow an appropriate expansion of their role. If that's done, these activities we're discussing can come about because they're logical extensions of the training that respiratory therapists have received.

Durbin: I'd like to leave one idea on the table. Should we require respiratory therapists to complete ACLS training? You mentioned that in your talk. Should that be a position of either the educational components or professional components of the AARC? I personally believe we should. That would give us much firmer ground to discuss these issues with other care providers who do require ACLS training.

Kacmarek: Well, let me answer that two ways. Let me answer it first from a practical perspective. We've tried to do that with our staff, and we have access to ACLS training programs; however, we're in the third tier of access. The physician, of course, is primary; the nurse is secondary; and we're on the tertiary level. Frequently, I can't get anybody into a program or at best one or maybe two of our staff. Access to training is one of the limiting factors. I think it could be better handled if it were one of the components of the formal training program—all therapists come out certified in ACLS. Every graduate therapist would be prepared. That's the only feasible way I see to accomplish universal training, at least on an initial start-up basis. Dean?

Hess: I think—and I would agree with Charlie (Durbin)—I think it's absolutely essential that therapists, particularly therapists who are responding to codes and are working in ICUs, be ACLS trained. I know that the therapists at our place have complained bitterly about going through those courses. They complain that they have to learn a lot of pharmacology and things that are very peripheral to their practice. But getting back to some of the points that you were making, Bob (Kacmarek), I think that if we as therapists are going to have any credibility at all in those environments, we have to have similar training. I don't think that we as therapists can ask to have a role in the decisions that are being made, to have a role in airway management (particularly intubation), to be doing defibrillation, and so forth, if we haven't acquired similar skills.

Kacmarek: Agreed.

Giordano: I want to give the perspective of the AARC with regard to the ACLS training. First of all, I certainly agree with the comments that qualified practitioners should take the course. Over the last 8 or 9 years, the AARC has encouraged access to those courses on behalf of the practitioners. We do have representation on the appropriate committee of the American Heart Association, as a matter of fact, and have had for about 7 or 8 years. It's my understanding that now, 8 years after we began the dialogue with the Heart Association, in the majority of states (and the decision on admission does reside at the state level) individuals who have at least a certification credential can go through ACLS training. About its being mandatory, I personally feel that it's now time to make it mandatory. We have been encouraging. It's now time to say, "OK, this is mandatory if you want to participate in the arena of emergency medical support."

Rodriguez: As a respiratory care manager, I have noticed that groups compete more successfully for scarce hospital resources when their profession has a well-established position or standard they can cite. The accessibility of ACLS training in hospitals can be very competitive. I, therefore, support Dr Durbin's suggestion that the AARC formally adopt the position that ACLS training is essential for respiratory care practitioners.

Kacmarek: I think most of us agree that there is a need for development of professional standards in this area.

Upper Airway Problems

Roger S Wilson MD

Introduction

Elective, emergency management of upper airway problems is accomplished routinely in operating and emergency rooms, critical care units, and prehospital settings. One single therapeutic maneuver, intubation, is performed by physicians and nonphysicians with varying levels of training and experience. This procedure is generally accomplished without difficulty, although it clearly has the potential for significant morbidity and mortality. It is important to recognize that adverse outcomes associated with respiratory events comprise the largest class of injury associated with the practice of anesthesiology, and airway management (including endotracheal intubation) is the most common single element.[1] Less certain is the frequency at which improper or inadequate approaches to airway management produce adverse outcome in patients treated in critical care, trauma, and cardiopulmonary resuscitation situations.

Failure to accomplish elective endotracheal intubation can be attributed to a number of variables including inadequacy of training, unsuspected anatomic and/or pathologic findings, and lack of adequate preparation for dealing with unavoidable difficulties. As previously stated, inappropriate airway management (including inability to efficiently perform endotracheal intubation, inadequate ventilation, and unrecognized esophageal intubation) is the leading cause of morbidity and mortality in hospitalized patients.[2] It is estimated that in the developed world approximately 600 deaths annually result from complications occurring at the time of intubation.[3] It is also evident that the incidence of difficulty varies with patient category; for example, the obstetric population is generally considered to be at much higher risk for intubation-associated problems than are other groups requiring such therapy.[4]

An individual's ability to intubate with a high degree of proficiency depends on that individual's knowledge of anatomic features, familiarity with equipment, and adequate practical ('hands-on') experience in a variety of settings. More important, however, is the intubator's ability to assess the situation appropriately, to develop an adequate plan or mental algorithm to effectively manage the dangerous and frequently unanticipated problems, and to provide a range of alternate therapies. It must be stressed that guidelines are often designed to fit the specific clinical context in which they are likely to occur, whether the in-hospital or out-of-hospital setting, and are influenced by the background and experience of those performing the procedures. The guidelines presented here are neither an exhaustive consideration of all possible conditions and manifestations of airway difficulty encountered nor all potential approaches to management possible for successful outcome.

Basic Concepts of Management

Anatomy

Knowledge of the anatomic features of the upper airway is essential for any individual—physician,

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Dr Wilson is Professor of Anesthesiology, Department of Anesthesiology and Critical Care Medicine, Memorial Sloan-Kettering Cancer Center and Cornell University Medical College, New York, New York.

A version of this paper was presented by Dr Wilson on October 3, 1991, during the RESPIRATORY CARE Journal Conference on Emergency Respiratory Care held in Cancun, Mexico.

therapist, or technician—who attempts endotracheal intubation. Figure 1, a sagittal view of the upper airway,[5] shows the important relationships among critical structures such as the epiglottis, vallecula, cartilaginous bodies, and vocal cords. A more detailed view (Fig. 2) depicts the relationship of laryngeal structures as viewed by the endoscopist during standard direct laryngoscopy. It is important to appreciate the relationships among these structures and the normal variations in individual components (such as size and shape of the epiglottis) prevalent in the population. Detailed information concerning anatomy and functional relationships is readily available.[6]

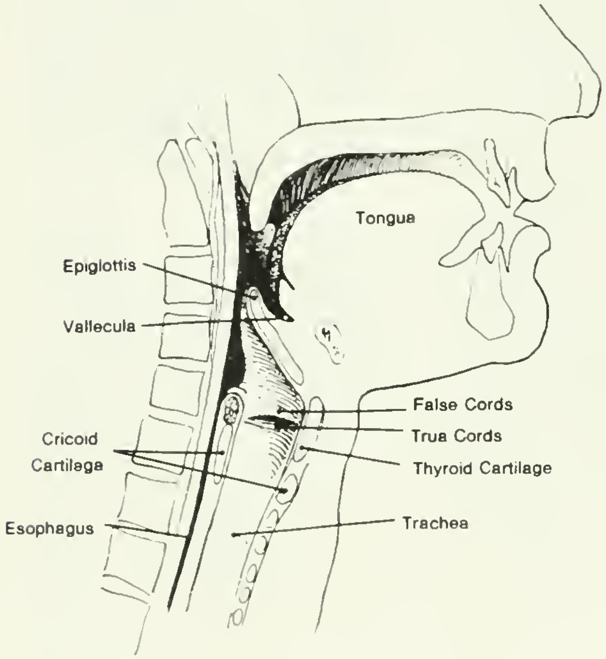

Fig. 1. Sagittal view of the head showing the anatomy of the upper airway. Note the anatomic relationships among the epiglottis, vallecula, cricoid cartilage, and vocal cords. (Reprinted, with permission, from Reference 5.)

Assessment

Assessment of the upper airway prior to intubation is a fundamental element in the approach to airway management. Because it is frequently the unanticipated difficult intubation that leads to a catastrophic outcome, it is important to review certain assessment techniques that have been advocated through the years. In the presence of obvious gross pathology involving soft-tissue and bony structures of the face and abnormalities of the neck and pharyngeal or laryngeal anatomy, one is alert to possible difficulties associated with laryngoscopic visualization and intubation. However, patients may appear to be quite 'normal' on casual visual inspection and yet present major difficulties during laryngoscopy and intubation. A number of methods have been advocated to alert the operator to impending difficulties prior to performing any procedure involving the airway.[7,8] Assessment encompasses a wide variety of activities (Table 1). Certain characteristics of the oropharyngeal cavity, teeth, and general body habitus, and head, neck, and jaw motion have been described (Table 2) as important predictors of a "difficult intubation."[9] Many variations in normal anatomy predispose an individual to difficulties during airway management. Correct

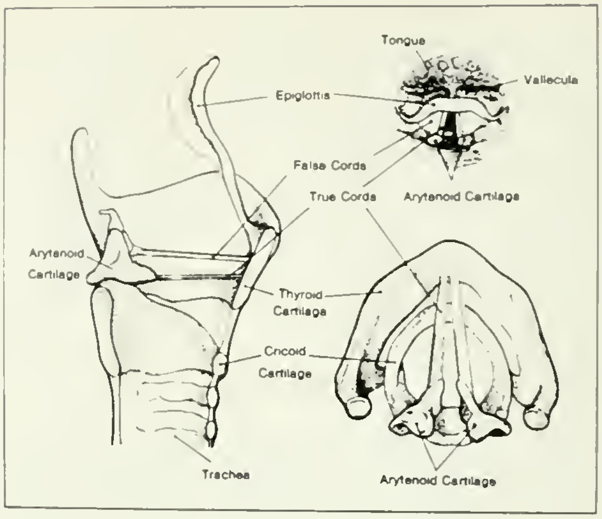

Fig. 2. Left, sagittal view of the upper airway. Top right, endoscopist's view of the larynx during laryngoscopy. Lower right, view of larynx with soft tissues removed. (Reprinted, with permission, from Reference 5.)

Table 1. Patient Characteristics To Be Assessed during Clinical Airway Evaluation

Body weight
Jaw movement
Head and neck movement
Thyromental distance
Oropharynx
Configuration of mandible and teeth

Table 2. Characteristic Features of the Patient in Whom Intubation May Be Difficult

Short muscular neck with a full set of teeth
Receding lower jaw with obtuse mandibular angles
Protuding upper incisor teeth with relative overgrowth of the premaxilla
Poor mobility of the mandible
Long, high-arched palate associated with a long, narrow mouth
Increased alveolar-mental distance necessitating wide opening of the mandible for laryngoscopy
Small glottic opening
History of tracheal stenosis
Poor mobility of the cervical vertebra

positioning of the head and neck to align the three major axes is of utmost importance in preparation for laryngoscopy (Figs. 3A-3C).[10] As described by Horton et al,[11] this position of flexion of the lower portion of the cervical spine combined with extension of the head at the atlanto-occipital joint should produce a lower-neck flexion angle of 35° and an extension of the facial plane at 15° relative to the horizontal plane. Inability to extend the head at the atlanto-occipital joint can result in conditions that allow less than optimal visualization of the glottis. The degree of extension possible at this joint is often a function of the abutment of the occiput against the posterior tubercle of the atlas. In general, the wider the atlanto-occipital distance in the neutral position, the greater the degree of extension that can be achieved. This distance or gap has been assessed by use of the lateral x-ray of the cervical spine in the neutral position. Several published reports have confirmed the value of this assessment in predicting difficulties during intubation.[12,13] Although there is wide variation in the atlanto-occipital distance when the head is in the neutral position, the presence of a narrow gap should cause concern.

In addition to normal variations in anatomy, other pathologic conditions of the cervical spine (including rheumatoid arthritis and calcification and bony protuberances) can produce a limited range of motion. In routine cases, it may be impractical to obtain radiographic assessment, and thus visual inspection of neck flexion and extension in the awake patient is of utmost importance.

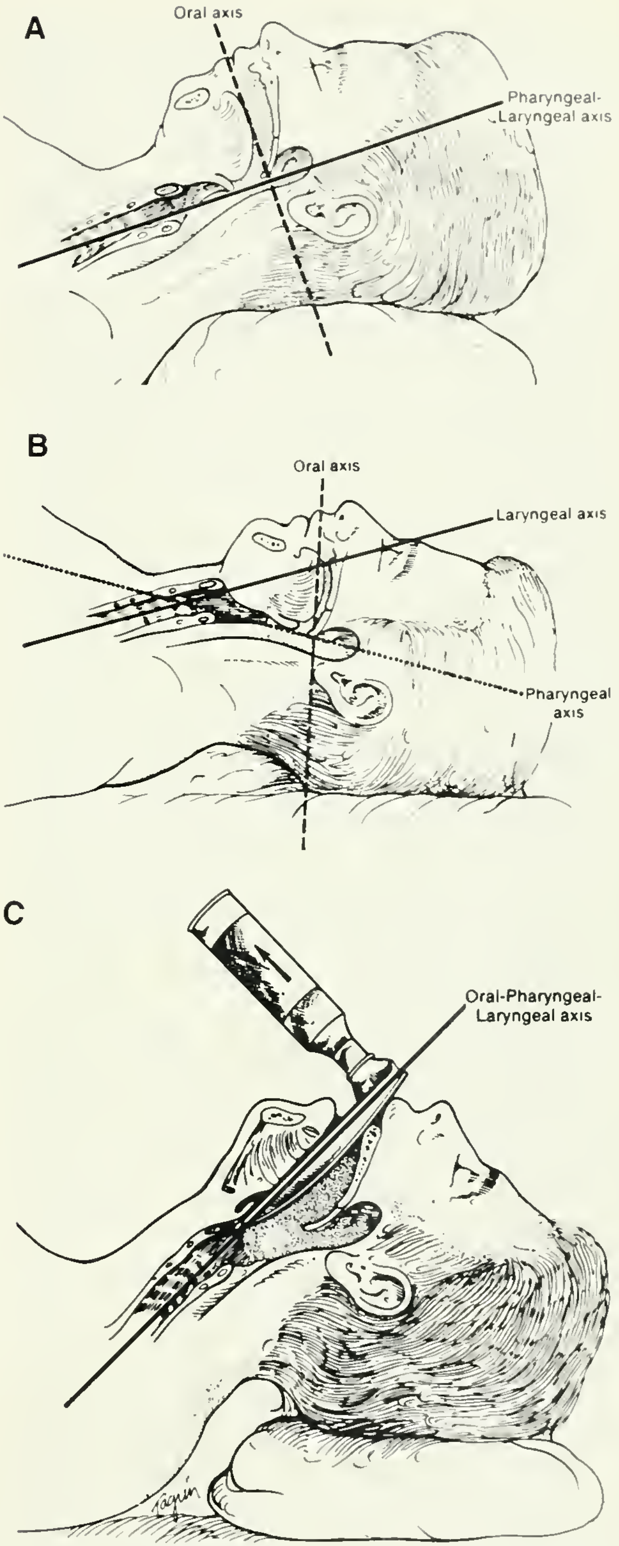

Fig. 3A-C. When the head is firmly supported and placed in the appropriate sniffing position, the axes align in one plane to facilitate laryngeal visualization. (Reprinted, with permission, from Reference 10.)

Wilson et al[14] identified and prospectively evaluated five other factors to determine their predictive value for difficulty encountered during subsequent intubation. Included were body weight, head and neck movement, jaw movement, the presence of a receding mandible, and the presence of 'buck' teeth. These factors (individually graded at three levels of severity) were prospectively tested using a group of 778 patients intubated by a group of 8 experienced anesthesiologists in an operating-room setting. As in most such published studies, prediction of difficulty based on such criteria is not always 100% accurate. In the study by Wilson et al, depending on the threshold level chosen, 75% of the laryngoscopies rated as difficult by the intubating physician would have been predicted by preoperative assessment; the false-positive rate was approximately 12%. When individual components—such as mandibular configuration, dental configuration (especially protruding upper teeth), and size of mouth opening—have been considered, the results confirm lack of uniformity in predicting the degree of difficulty encountered during intubation.[7] It is important to underscore the message that although visual inspection and assessment of surface anatomy and motion prior to airway management are well described, routinely practiced, and important, they are less than totally accurate in predicting difficulties.

Mallampati et al[15,16] and somewhat later Samsoon and Young[17] attempted to correlate clinical observation of certain soft-tissue structures within the oropharynx to the degree of visual difficulty during laryngoscopy. In theory, when the base of the tongue is unusually large it results in poor visualization of faucial pillars, uvula, and soft palate. These findings are potential predictors of inadequate visualization of laryngeal structures during laryngoscopy. Preintubation assessment is performed with the awake patient sitting in the upright posture with head in a neutral position. The mouth must be opened as wide as possible and the tongue extended to permit the observer, sitting at eye level, to inspect the pharynx. Mallampati and colleagues[15,16] originally grouped patients into one of three classes based on whether, during preintubation inspection as described, all structures, some structures, or only the soft palate could be visualized. This classification system was prospectively studied in 210 patients to confirm its clinical utility. Results, summarized in Table 3, compare preintubation classification with degree of difficulty encountered by physicians and nurse anesthetists.

Table 3. A Scheme for Predicting Intubation Difficulty from Preintubation Inspection*

Preintubation Classification	Difficulty Encountered during Intubation			
	Grade 1[†]	Grade 2	Grade 3	Grade 4
Class 1[‡] 155 (74%)[§]	125 (59%)	30 (14.3%)	0	0
Class 2 40 (19%)	12 (5.7%)	14 (6.7%)	10 (4.7%)	4 (1.9%)
Class 3 15 (7%)	0	1 (0.5%)	9 (4.3%)	5 (2.4%)

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[*] Adapted from References 15 and 16.

[†] Grade 1 (all glottic structures visualized during laryngoscopy) progressing to Grade 4 (no glottic structures including corniculate cartilages visualized).

[‡] Class 1 = all pharyngeal structures visualized; Class 2 = some pharyngeal structures visualized; Class 3 = only soft palate visualized.

[§] Number of patients (% total).

Grade 1 conditions were described as allowing visualization of all glottic structures, whereas the other extreme, Grade 4, described circumstances in which no glottic structures including corniculate cartilages could be seen during standard laryngoscopy. As described in the table, Class-1 patients were always within Grade 1 or 2 (ie, adequate exposure during direct laryngoscopy), whereas the preponderance of Class-3 patients fell into Grades 3 and 4 (ie, inadequate exposure). Class-2 patients demonstrated considerable overlap between grade levels and, although the results are highly significant and support the hypothesis that difficult laryngeal visualization can be predicted, application of the system does not predict difficulty with 100% accuracy. Other generalized conditions such as edema occurring with pregnancy, organ system disease, or local infection and/or trauma may confound predictability when soft-tissue configuration is being evaluated.

Finally, simple observations have been described as predictors of difficulty. White and Kander[18] have noted that an increase in the posterior depth of the mandible can limit displacement of soft tissues by the laryngoscope blade and thus limit visualization. Measurement of the distance from the thyroid notch to the mental protuberance with the neck extended can be useful.[19] Reduced mandibular length and increased anterior depth of the mandible have also been found in patients judged difficult to intubate.[20,21] Measured distances such as between the mentum and thyroid cartilage are likely to be influenced by anatomic factors such as laryngeal position, obesity, and head extension as well as the already described characteristics of the mandible. Although the features described (either alone or in combination) have predictive value, no one single observation has proved to be superior. However, such observations do, with minimal commitment of time and effort, increase the awareness of difficulties that may be encountered.

Upper Airway Pathology

In addition to variations of normal anatomy, pathologic conditions must be considered. Four representative types of pathology with the potential for complicating laryngoscopy and intubation are summarized in Table 4.

Table 4. Upper Airway Pathology that May Make Intubation Difficult

Congenital defects
Edema
post-traumatic
infectious
Neoplasm
Trauma
acute
delayed (stenosis)

Congenital Lesions

Congenital lesions are a consequence of abnormal embryologic development and range in complexity from severe, life-threatening, and obvious defects to those that are much more subtle and indistinct. Pathology can exist at any site within the upper respiratory tract including the nasopharynx, larynx, and upper trachea. Obvious craniofacial defects include congenital fusion of the jaws, reported to frequently occur in association with aglossia (absence of the tongue) and retrognathia (presence of a receding jaw). Macroglossia, with or without associated lymphangioma or hemangioma, makes direct visualization of laryngeal structures unlikely. More complicated defects such as maxillofacial cleft, mandibulofacial dysostosis (Treacher-Collins syndrome), craniofacial dysostosis, and Klippel-Feil syndrome, and the problems that they pose during airway management have been well described.[22] These and other soft-tissue, osseous, and cartilaginous malformations, usually evident on clinical examination, are examples of congenital lesions that should raise awareness and concern prior to intubation. Less obvious, but equally important, are congenital lesions involving the supra- and subglottic area, including cysts and cystic hygroma. Such lesions may not be noted during clinical evaluation due to their subtle presentation, but they can produce anatomic obstruction, interfere with ventilation during induction, and create difficulty during intubation. More distal congenital vascular lesions including aberrant pulmonary and innominate arteries and double aortic arch create special problems. These lesions are not obvious during clinical evaluation or in the course of laryngoscopy but rather can impede distal placement of the endotracheal tube into the airway once it passes through the vocal cords. Congenital stenosis in the immediate subglottic area or in the more distal trachea can produce similar problems. Although many of these conditions result in unavoidable problems during airway management, a high index of suspicion and a well-executed alternative plan are essential for a desirable end result.

Edema

Two common mechanisms of edema production are airway trauma and infection. Edema can result from direct, blunt trauma of the face and neck or recent therapeutic or diagnostic airway manipulation (eg, laryngoscopy, bronchoscopy, or endotracheal- or nasogastric-tube insertion). Recognition and anticipation of difficulty in the presence of edema are important because any additional trauma from airway manipulation can further compromise an already tenuous situation. Conditions can quickly worsen to make ventilation impossible and necessitate an invasive approach to secure the airway. Inflammatory causes of edema encompass a number of bacterial and viral infections. Supraglottic structures are frequently involved as a result of bacterial infection. Retropharyngeal and tonsilar abscess have the potential for markedly altering the anatomy of the upper airway causing airway obstruction and producing conditions that hinder intubation by standard techniques. In addition, epiglottitis (generally secondary to *Hemophilus influenzae* or staphylococcal or streptococcal infection) may produce a rapidly progressive, supraglottic swelling. This clinical entity must be cautiously managed by the most experienced endoscopist available because measurable morbidity and mortality exist when instrumentation is performed under uncontrolled circumstances. Viral infections can result in lymphadenopathy and localized subglottic edema. Croup, secondary to a localized viral infection, results in an edematous condition that reduces cross-sectional diameter of the airway in the subglottic area. Any traumatic or uncontrolled attempt at placement of an endotracheal tube through an airway with pre-existing edema and limited cross-sectional airway diameter can, if unsuccessful, result in rapid progression of swelling and resultant total airway obstruction.

Neoplastic Lesions

Benign and malignant tumors can involve the airway at various levels including the oral cavity, posterior pharynx, larynx, and trachea. A variety of primary and secondary (metastatic) lesions have been described within the airway.[23,24] Airway obstruction, inability to visualize laryngeal structures due to mass effect in the oral or posterior pharynx, and/or inability to pass endotracheal tubes through laryngeal or tracheal structures are potential problems. These lesions create a potentially complex situation because bleeding, fragmentation of the tumor mass (especially where it is necrotic and friable), and swelling secondary to direct trauma may create unpredictable and life-threatening situations. Recognition, rapid action, and a well-thought-out alternative approach to airway management are essential to effective handling.

Traumatic Injury

Traumatic injury most commonly involves the airway at the level of the larynx, cervical trachea, or carina. Acute trauma, secondary to blunt injury to the face and neck, can create a circumstance in which a functional airway exists and allows adequate gas exchange but also a circumstance in which attempts at intubation with resultant airway dehiscence may accentuate the injury, with consequent morbidity and mortality. Airway management under these circumstances relies on experience, versatility of approach, and a coordinated effort by those providing therapy.[25]

Delayed, postintubation, and/or post-tracheotomy lesions generally are stenotic or malacic. Stenosis secondary to ulceration and scar formation generally involves supra- and subglottic areas and the upper trachea. Factors such as tube size, cuff design, duration of intubation, local infection, and generalized state of nutrition govern the development and extent of injury.[26,27] These specific examples of airway pathology are important because they represent clinical situations that require appreciation of etiology of injury, an organized approach to management, and an alternative method when the primary technique fails.

Intubation Techniques and Equipment

Equipment

Standard direct laryngoscopy utilizes a laryngoscope comprised of a self-contained battery handle and one of a wide variety of curved or straight blades. Advantages and disadvantages, appropriate positioning, and the technique of rigid laryngoscopy are well described.[28,29] Fiberoptic endoscopy utilizes malleable and flexible endoscopes designed specifically for laryngoscopy and endotracheal intubation. The third technique includes retrograde translaryngeal wire-guided intubation and use of illuminating intubation stylets or wands. Each of these techniques requires adequate experience, complete understanding of the technical approach, and a knowledge of specific advantages and disadvantages of each.

Technical Approach

Three major categories of approach to elective and emergency endotracheal intubation can be described. The direct approach, using one of a number of standard rigid and fiberoptic laryngoscopic techniques, involves direct vision of the laryngeal structures and subsequent intubation. Indirect methods, frequently designated "blind intubation," usually involve tube placement without visualization of airway structures, with position governed by breath sounds and patient response. A third, invasive approach, involves transcutaneous cannulation of the trachea or placement of a wire or stylet for retrograde intubation. Level of patient awareness varies from awake to completely anesthetized, and muscle relaxants may or may not be used.

A specific strategy with alternative measures is an important consideration when difficulty in airway management is encountered. The degree of benefit derived from a planned approach, although not proven, is suggested in the literature. Specific strategies are often presented in the form of a comprehensive plan or algorithm as shown in Figure 4.[30] Although a variety of algorithms have been advocated and published, various factors influence the form they should take. Factors include types of patient managed, anticipated skill and experience of those providing care, and, potentially, the setting (in- or outside of the hospital, operating suite, ICU, or other urgent-care location).

The general strategy should consider patient awareness first. In situations in which a difficult intubation is anticipated (eg, prior history of difficulty, obvious anatomic abnormalities, or trauma), the initial approach should incorporate use of topical anesthesia and, if feasible, local nerve blocks. Sedation is often utilized once the operator has assessed patient response, level of discomfort, and the technical aspects of airway management Caution is necessary at this stage because a poorly thought-out or mismanaged pharmacologic approach can convert a stable situation to one of difficulty. This may occur secondary to inexperience, poor selection of drug and dosage, or inability to appropriately assess airway conditions.

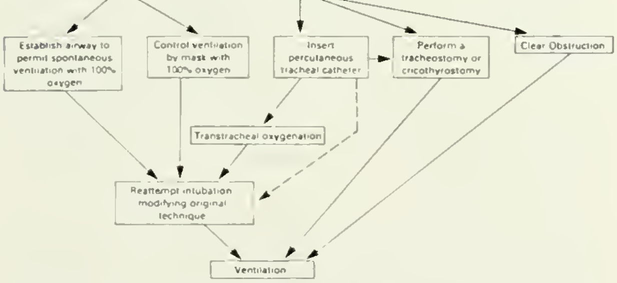
Intubation attempt fails
Patent airway
Obstructed airway
Establish airway to permit spontaneous ventilation with 100% oxygen
Control ventilation by mask with 100% oxygen
Insert perculaneous tracheal catheter
Perform a tracheostomy or cricothyrostomy
Clear Obstruction
Transtracheal oxygenation
Reattempt intubation modifying original technique
Ventilation

Fig. 4. Flow chart for airway management when attempted intubation is unsuccessful. (Reprinted, with permission, from Reference 30.)

Blind Intubation

Blind nasal intubation is advantageous for the patient who is awake and breathing spontaneously and, in general, is an ideal choice when time is not an important factor. Preintubation preparation with use of topical anesthesia and a vasoconstrictor (cocaine or a phenylephrine) in the nasopharynx is important to ensure patient comfort and cooperation. Selection of an endotracheal tube of appropriate size is most important. Placement is governed by visual and tactile cues, including soft-tissue resistance with tube advancement, air movement, and, frequently, evidence of condensation during exhalation on the inner wall of the endotracheal tube. Coughing, normalization of inspiratory and expiratory efforts, and increased airflow are often end points of successful tube placement. Advantages of this approach are that the patient continues to breath spontaneously, has ability to clear secretions from his airway, and can be managed in a variety of positions. Disadvantages include the fact that it is time-consuming and, when improperly performed, may produce undue trauma to the nasal mucosa and/or structures of the posterior pharynx. The diameter of the tube used is limited by the size and shape of the nasal passage; a longer tube is required, and complications including sinusitis and necrosis of the nares are possible.

Awake Oral Intubation

Awake oral intubation is an approach that requires adequate time to establish appropriate topical anesthesia of oral and pharyngeal structures and/or use of nerve blocks. When done under direct vision, some degree of sedation is generally required because placement of a foreign body such as a laryngoscope blade in the posterior pharynx produces a strong stimulus for gagging, coughing, and movement. A disadvantage is the potential need for significant amounts of sedation resulting in loss of cooperation and airway protective reflexes. Advantages over the nasal approach include a decreased risk of soft-tissue trauma, use of a tube of larger bore and shorter length, and avoidance of complications such as sinusitis and middle-ear infection.

Flexible Fiberoptic Intubation

Flexible fiberoptic endoscopy for intubation, described by Murphy[31] in 1967, has become an important adjunct in management of normal and abnormal airways (Table 5). This approach is a useful alternative to performing conventional nasal or oral intubation in awake or minimally sedated patients. It allows more complete visualization of airway structures than may be possible by conventional laryngoscopy in some patients. In addition, it is less stimulating and thus more comfortable than is rigid-blade laryngoscopy when correctly performed. Limited operator experience and the presence of abundant secretions and excessive quantities of blood in the airway can complicate implementation. Secondary advantages include ability to suction secretions from the airways during intubation, capability to administer supplemental oxygen during the procedure, and ability to confirm intubation and tube position (particularly important when unusual tube positioning, such as selective endobronchial intubation or tracheal pathology, requires precise placement). A number of excellent reviews and monographs have detailed the specific details for implementing fiberoptic techniques.[32-34]

Table 5. Indications for Fiberoptic Intubation

- Routine intubation (experience/teaching)

- Difficult intubation
  - Anticipated
    - History of prior difficulty
    - Physical evidence of difficulty
  - Unanticipated
    - Failure of standard methods

- Compromised airway
  - Upper airway pathology
  - Tracheal pathology

- Limited neck mobility
  - Trauma
  - Chronic disease

Rigid Fiberoptic Intubation

Rigid fiberoptic laryngoscopes, similar in design to rigid bronchoscopes, are suitable for an oral approach only and require an experienced operator to accomplish 'straight line' introduction into the upper airway. Because of the rigid design, the patient's anatomy must conform to the configuration of the scope, and management of jaw and tongue position is critical.

So-called malleable fiberoptic laryngoscopes offer advantages and disadvantages of both flexible and rigid designs. They are 'molded' to conform to the anticipated shape of the patient's airway prior to insertion. Passage through the oral and posterior pharyngeal cavity is less difficult than with a truly straight rigid scope and somewhat easier to direct than with a truly flexible device, which requires somewhat more precise control. The malleable scope has the advantage of being able to move soft tissue out of the way during advancement. In addition, the instrument, manufactured by the American Optical Company, can be illuminated by a battery in the handle or by a cable and standard high-intensity light source.

Retrograde Techniques

Retrograde intubation techniques are useful in many special circumstances especially in patients who have sustained significant maxillofacial trauma.[35] The method of choice is determined by clinical circumstances, availability of equipment, and experience of the operator. A number of retrograde techniques have been described that use guides that pass percutaneously through the cricothyroid membrane and exit via the mouth or nares.[36,37] Guides have included small venous and epidural catheters, vascular wires (Seldinger or J-wire), and silk and nylon thread.[38,39] A simple approach utilizes readily available vascular-access equipment consisting of a 19-gauge thin-wall needle and J-wire. Following appropriate positioning and skin preparation, the needle is passed percutaneously through the cricothyroid membrane and directed approximately 30° cephalad from the perpendicular. The guide is advanced retrograde into the airway and through the larynx to enter the oral cavity or exit via the nares. An appropriately sized endotracheal tube is threaded over the guide and advanced into the upper airway and the laryngeal aperture.[40,41] Difficulty can occur when passage of the tip of the endotracheal tube into the larynx is attempted and is diverted by soft-tissue structures such as the epiglottis or vallecula. Tube rotation, movement of the larynx by the operator via the anterior surface of the neck, or use of larger, stiffer guides and fiberoptic bronchoscopes passed antegrade over the original guide are used to resolve this problem.[42,43] Although no data are available to determine the success rate of this approach, it is an important and perhaps underutilized technique for the management of difficult airways.

Illuminated Wands

Light wands or illuminated stylets have been successfully used to facilitate tube entry into the larynx without direct vision.[44,45] The device is passed in awake or anesthetized patients via nose or mouth, and transillumination aids in the identification of relevant structures. This relatively simple yet efficient technique has been shown to be effective both in adult and pediatric patients,[46,47] and is a logical alternative when conventional approaches have failed. One specific application is in cases in which cervical-spine pathology limits proper neck motion and head position.[48] Effective use of this technique can be limited by lack of appreciation for anatomy, excessive quantities of subcutaneous tissue, and bright levels of ambient light.

Airway Management without Intubation

In special circumstances, intubation may not be possible and mask ventilation may not maintain adequate gas exchange. This probably occurs most frequently in the prehospital setting when care providers are not suitably trained or permitted to intubate. Alternative techniques have been developed to fill this therapeutic void. In addition, some in-hospital patients cannot be intubated by conventional techniques because of upper airway pathology.

Esophageal Obturator Airway (EOA)

This device (Fig. 5[49]) was described by Don Michael et al[50] in 1968, and has been used extensively in clinical practice since 1972. The tube with occluded distal tip is introduced blindly into the mouth and then advanced into the esophagus. Once in position, the cuff is inflated and the mask positioned appropriately over the mouth and nose. Bag-to-tube or mouth-to-tube ventilation escapes via the small holes in the tube into the pharynx and subsequently passes through the larynx into the lung. The balloon and occluded distal tip prevent air from entering the stomach and protect against gastric regurgitation. Adequacy of ventilation depends on a secure mask fit, appropriate balloon inflation, and patency of the larynx and conducting airway.

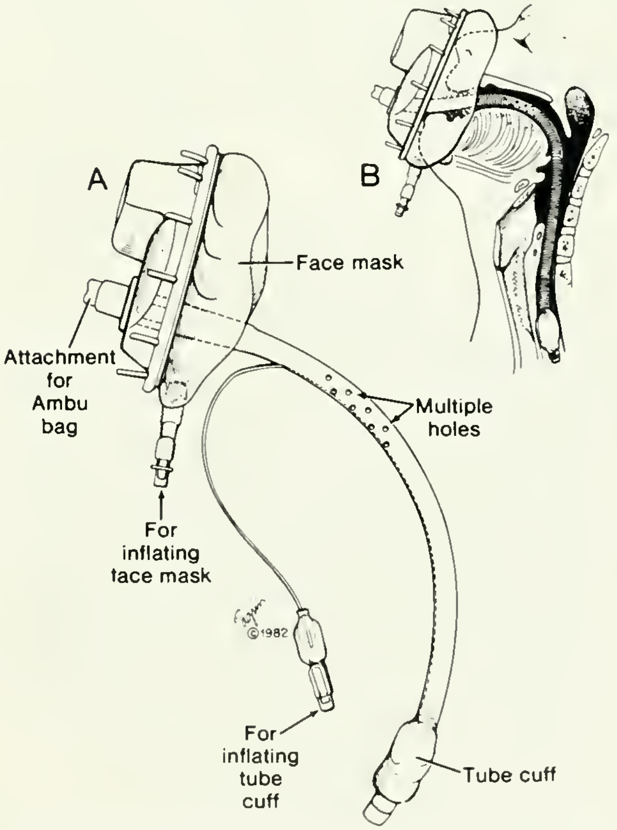

Fig. 5. Esophageal obturator airway. See text for details. (Reprinted, with permission, from Reference 49.)

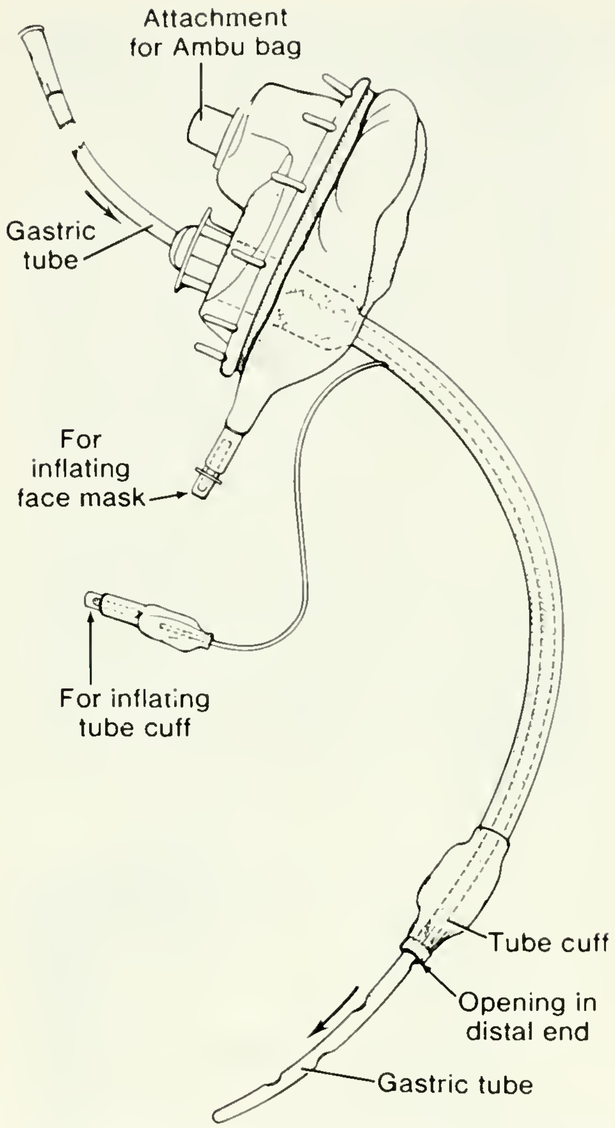

Fig. 6. Esophageal gastric tube airway. See text for details. (Reprinted, with permission, from Reference 49.)

The principal use of the EOA has been by paramedics in the setting of prehospital cardiopulmonary resuscitation, with general overall success.[51,52] Reported complications include technical difficulty converting to standard intubation, esophageal injury, and inadvertent tracheal intubation.[53] The role of EOA in the context of unsuccessful intubation in hospitalized patients is probably minimal given the availability of better alternatives.

Esophageal Gastric Tube Airway (EGTA)

The application and utility of this device (Fig. 6) are similar to the EOA. The major difference is the patent lumen of the esophageal tube that permits gastric decompression. The gas-exchange port is located on the mask. Factors governing inadequate function and complications are similar to those cited for the EOA.

Esophageal Tracheal Combitube (ETC)

This device (Fig. 7) combines the features of an esophageal obturator and an endotracheal tube. As described by Frass et al,[54] the ETC is a twin-lumen tube with one lumen resembling an EOA and the other an endotracheal tube. A pharyngeal balloon, similar in function to the mask of an EOA, prevents egress of gas through the mouth and nose. A smaller cuff at the distal end of the tube serves to seal either trachea or esophagus depending on location. Perforations in the esophageal lumen provide ventilation via the pharynx through the larynx in a fashion similar to the EOA if the tube is passed blindly into the esophagus; if placed into the trachea, the patent tracheal lumen is utilized for ventilation in a conventional manner. The versatility provided with the double-lumen system obviates some of the potential hazards described with other esophageal tubes. Adequacy of function has been described during conditions of cardiopulmonary resuscitation, surgery,[54] and intensive care.[55] The full extent of clinical application both in and out of hospital settings remains to be defined.

Laryngeal Mask Airway (LMA)

This oval mask-like device (Fig. 8), originally described by Brain in 1983,[56] was designed to be

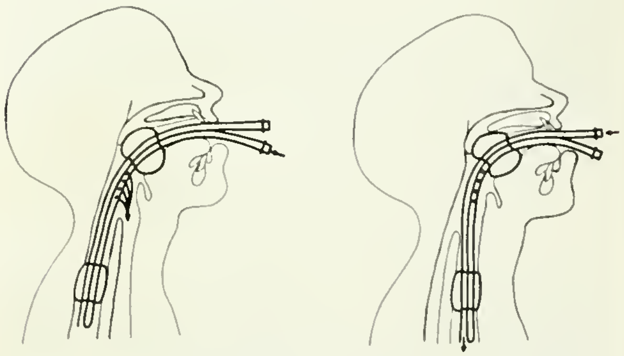

Fig. 7. Esophageal tracheal Combitube. (Reprinted, with permission, from Reference 50.)

inserted into the posterior pharynx to provide an airway in anesthetized patients. The mask with an inflatable rim is connected to a 12-mm-ID tube. With the patient's head in a sniffing position, the lubricated mask is placed into the oral pharynx with the mask opening lying adjacent to the tongue. The mask is advanced until resistance is encountered, which generally means that the tip rests in the proximal esophagus with mask opening adjacent to the larynx. Once in position, the rim cuff is inflated to produce a seal. Tube and laryngeal mask allow gas flow through the nonintubated larynx. The utility of this device lies between those of mask ventilation and tracheal intubation. Advantages are associated with ease of placement by trainees with minimal experience when compared to attempted endotracheal intubation.[57] Potential difficulties include obstruction of the laryngeal aperture, laryngospasm, and aspiration (the latter possible because the esophagus and larynx are not isolated with this technique). The role of this seemingly simple device in the prehospital setting and in management of patients with airway pathology, failed intubation, and other conditions remains to be defined.

Transtracheal Jet Ventilation (TJV)

This is an invasive, potentially life-saving procedure suitable for the patient who is impossible to intubate and is difficult to ventilate with standard noninvasive approaches. As extensively reviewed by Benumof and Scheller,[58] TJV has been shown to be a relatively safe, extremely effective mode of therapy in situations in which ventilation and intubation are not possible. Percutaneous TJV when performed with a large-bore I.V. catheter or similar device is much more expedient than a more traditional tracheotomy and perhaps even percutaneous cricothyroidotomy. A number of variations of system design have been described.[58] A shortcoming of this technique is that all of the appropriate equipment including catheters, connectors, and a high-pressure gas source must be readily available at the time and place that airway difficulties are encountered. Serious complications of TJV are essentially those related to delivery of gas under high pressure and needle-puncture injury. Most serious are subcutaneous emphysema, mediastinal emphysema, pneumothorax, esophageal puncture, bleeding, and hematoma.[59,60] In general, such complications can be avoided with appropriate patient selection and careful and correct placement of the catheter into the airway to ensure adequate egress of air from the respiratory system when the catheter is connected to a high-pressure gas source.

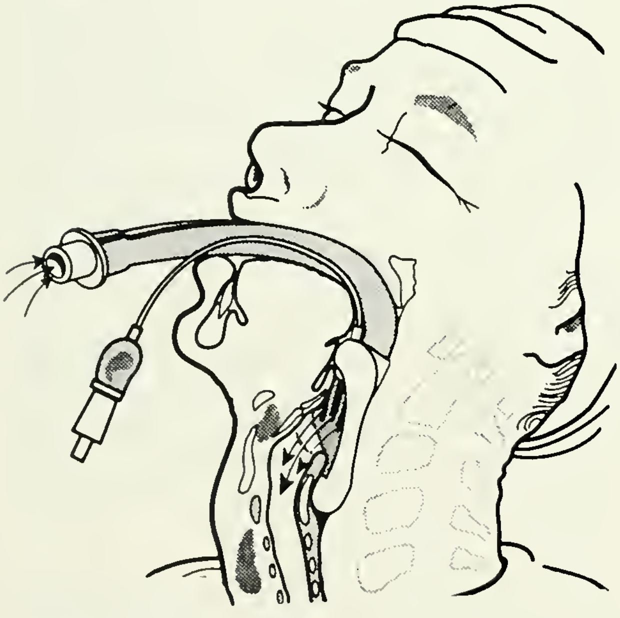

Fig. 8. Laryngeal mask airway. (Reprinted, with permission, from Reference 53.)

Minitracheotomy

Self-contained kits provide small-bore catheters with appropriate adapters to facilitate gas flow in those patients who are unable to ventilate and cannot be intubated. Although many such kits were designed to provide airway access for suctioning in the in-hospital patient, they can provide rapid access into the airway via the cricothyroid membrane.[61] An advantage of this system is that all necessary equipment is readily available in one kit and adapters allow I.V. catheters to be attached to conventional hand resuscitator systems to facilitate ventilation. Complications of this technique are similar to those described for TJV.

Pharmacologic Adjuncts to Airway Management

Pharmacologic intervention is often required to provide increased patient comfort and cooperation and to facilitate laryngoscopy and endotracheal intubation. A variety of agents are utilized including (1) sedative hypnotics (for decreased awareness and reduced level of consciousness), (2) narcotic analgesics (for decreased pain), (3) anesthetic agents (to produce a state of unconsciousness), and (4) muscle relaxants (to prevent movement and eliminate muscle tension). Drugs from several categories are often administered in combination depending upon the desired objectives. Although use of these agents clearly can facilitate both laryngoscopy and intubation, steps must be taken to ensure that they are titrated to the desired effect because each alone or in combination can have untoward effects that may compound an already existing critical situation. Avoidance of such complications depends upon a knowledge of the pharmacodynamics and pharmacokinetics of these drugs, appropriate selection in any given clinical situation, and ability to manage complications and side effects should they occur. I focus on specific agents most frequently used in the course of airway management and do not provide an exhaustive and authoritative review of the subject.

Sedative Hypnotics

Sedation may be produced by several types of drugs including barbiturates, benzodiazepines, and anesthetic-induction agents including ketamine and propofol. In general, the intent of therapy is to reduce anxiety while minimally affecting protective mechanisms such as gag, swallow, and coughing. Characteristically, this group of agents produces a spectrum of effects ranging from mild sedation to severe central nervous system depression and resulting coma. They provide no analgesia: patients often respond to painful stimuli as exhibited by movement and autonomic nervous system activity. Amnesia is governed by the specific agent used and dose administered. Barbiturates, especially thiopental, are often selected because their rapid onset and short duration of action are useful when given in appropriate amounts. These agents may, in higher doses, produce profound unconsciousness, loss of protective reflexes, and cardiorespiratory depression. Recovery is a function of redistribution and metabolism without availability of a specific 'reversal' agent.

Benzodiazepines including diazepam (Valium) and midazolam (Versed) have been widely used during therapeutic and diagnostic procedures involving the airway. Sedation with amnestic effect and titratable characteristics make these agents attractive for such applications. Cardiac depression related to alpha-adrenergic blocking effects of the drugs is an anticipated side effect; respiratory depression, especially when combined with narcotic administration, must also be anticipated. Duration of action is agent-specific, dose-dependent, and governed by other variables such as age. Again, experience and ability to manage the untoward effects are of utmost importance. Propofol, a new lipid-soluble isopropyl phenol produces a rapid induction of anesthesia, similar to barbiturates when given in suitable doses (1.5-3 mg/kg) and lasting for a period of minutes. When given in lesser concentrations, by slow I.V. titration, sedation without loss of consciousness is easily achieved. In contrast to the barbiturates, residual sedative effects are minimal upon awakening, making it an ideal agent for airway manipulation. Obtunded airway reflex and minimal side effects such as nausea and vomiting are marked advantages; respiratory and cardiovascular depression, although dose related, are undesirable side effects.

Ketamine, a phencyclidine derivative, produces a rapid onset of unconsciousness after intravenous doses of 1-2 mg/kg. Lower doses tend to produce a dissociative state without true unconsciousness but provide some amnesia and some analgesia. Persistence of skeletal muscle tone, especially in the upper airway, is a somewhat undesirable feature during airway manipulation. Cardiovascular function is well maintained due to direct stimulation of the sympathetic nervous system unless there is a pre-existent depletion of endogenous catecholamines. Emergence, especially if sufficient quantities are administered, may be associated with disturbing auditory, visual, and proprioceptive illusions that can lead to a state of delirium. With the advent of shorter acting benzodiazepines and drugs such as propofol, use of ketamine during airway manipulation has become less attractive.

Narcotic Analgesics

Several naturally occurring and synthetic narcotics are available for use during airway procedures.

These drugs, generally, are central nervous system depressants often producing a sense of tranquility and euphoria, decreased discomfort and awareness, and suppression of cough reflex.

The most common adverse side effects are profound respiratory and cardiovascular depression. Narcotics are most useful adjuncts when titrated in combination with other drugs such as barbiturates or, more commonly, the benzodiazepines. Dosage and duration of effect are a function of the specific agent selected. A decided advantage for use of these drugs for patient comfort is the availability of specific narcotic antagonists, such as naloxone, to reverse untoward effects. Severe cardiovascular depression, frequently due to histamine release and vasodilatation, must be considered.

Muscle Relaxants

Muscle relaxants function predominantly at the myoneural junction producing intense skeletal muscle relaxation of rapid onset and variable duration. Agents and their characteristics are detailed in Table 6. Use of muscle relaxants in the setting of airway pathology requires sound clinical judgment and a number of skills. Caution in producing respiratory paralysis cannot be overemphasized, and it is strongly recommended that these agents be administered only by those physicians who have substantial experience in mask ventilation, the standard approach to intubation, and the ability to undertake the special procedures as necessary.

In Summary

The appropriate management of patients with airway pathology represents a complex interaction among patient-specific factors, the clinical setting, and the knowledge and actions of the practitioner. Decisions must be made quickly, alternative approaches reviewed, and a plan of action undertaken in rapid sequence. Skill and experience (as well as ability to perform a variety of technical activities) are essential. This review serves only to outline those issues that must be considered and to provide a framework in order to develop the appropriate approach to the management of these extremely difficult problems.

Table 6. Muscle Relaxants Used during Intubation

Drug	Intubating Dose (mg/kg)	Onset Time (min)	Duration (min)	Fate	Side Effects
Succinylcholine chloride (Anectine)	1.0-1.5	0.5-1.0	10-15	Metabolism = blood pseudocholinesterase	Hyperkalemia, bradycardia, tachycardia, prolonged blockage
Pancuronium (Pavulon)	0.08-0.1	1-3	35-55	Metabolism = liver excretion via kidney/liver	Tachyarrhythmias, hypertension
Atracurium (Tracrium)	0.4-0.5	3-5	20-45	Metabolism = Hofman elimination/ ester hydrolysis	Histamine release at higher dosage
Vecuronium (Norcuron)	0.08-0.10	3-5	25-40	Metabolism = liver excretion via kidney/liver	No significant cardiovascular effects

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Wilson Discussion

Hess: Roger, I have a couple of questions. First, the use of a lighted stylet has been described in the emergency medicine literature,[1] and I wonder if you would comment on its use in prehospital management. Second, do you have any suggestions about when a cricothyrostomy should be converted to a more conventional tracheostomy? In our ICU, from time to time we have patients come up from the emergency department with a cricothyrostomy received in the field or the emergency department, and there seems to be some confusion in the minds of the staff regarding the need to convert to a more conventional airway.

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Wilson: One of the techniques that has been advocated is use of a flexible intra-operative light source or one of the new lighted stylets. Much of my personal experience has been under the circumstances of controlled or elective intubation, in which the stylets work very well—situations in which you can control the outside lighting, pick patients because of their anatomy (again, thin necks), and pick individuals in whom one can manipulate the anatomy. Under such circumstances (as borne out by the literature), lighted stylets work really well.[1] When you encounter situations in which you are dealing with individuals who have abnormal pathology, blood, and secretions; are moving around and coughing; and are not as cooperative as anesthetized individuals, the success rate tends to drop. I think it's a viable technique. I think the nice part of the technique is that it is easy to train individuals to use it, with essentially zero morbidity, because it can be done under controlled circumstances. It's not costly. The stylets are easy to stock in the emergency room. You have to use experience and good judgment as to when you think it's the next logical step, based on the situation and the urgency, to get the individual intubated. The second question is a bit more difficult to answer. What do you do once a cricothyroid membrane has been used as a point of entry for either a small endotracheal or tracheostomy tube? The answer depends on one's impression of the literature and what you base your decisions on with respect to the number of complications. Criothyrotomy was initially advocated as a very safe procedure when compared to a tracheotomy at the first or second ring.[2,3] The problem is in evaluating a series from one institution or series in which individuals have taken this as a pet project—they do it; they do it well; and they have a vested interest in the procedure. Subsequent series have not borne out the low incidence of complications. In fact, the problem with leaving this larger tube in through the cricothyroid, although the incidence of complications may be low, is that lesions that do result are not very reparable. They are very difficult lesions to fix. Often patients end up with airway appliances in for the rest of their lives, as opposed to pathology in the lower portion of the trachea, which, if diagnosed properly and managed well, is 'fixable' under almost 100% of circumstances as long as the surgeon knows what he is doing. So, the tendency, I think, is to move away from long-term use. Certainly it is an important portal for the airway in emergency situations, but, again, one that has been oversold in terms of its benign nature. Our experience with 'minitrachs,' placing a 6-mm catheter in the airway, even under controlled circumstances with a thoracic surgeon doing it, has been associated with 2 or 3 instances of really massive hemoptysis in about 100 placements (a topic which is dear to Dr Stoller's heart). Again, under best of circumstances, this is not absolutely benign territory to deal with; so, placement of needles, wires, small catheters, or anything else through this membrane should be done by people who are skilled and are knowledgeable, and, more importantly, can handle, on an urgent basis, the consequences of producing more problems.

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Stoller: My question regards proficiency with the airway—getting it and maintaining it—and bears a little bit on Bob's (Kacmarek) talk. The question really is, How steep is the learning curve to develop mastery over the airway with regard to routine kinds of situations, and then the more specialized, difficult circumstances you've described? What level of experience is required to acquire skills and to maintain these skills? I suspect there's not much in the literature, but the answers will impact greatly on the level of involvement of therapists in ongoing participation in airway management.

Wilson: It's a good question. I think the answer is very subjective because the data are not there. I think one is often biased by his own personal experience. The circumstance I would cite is dealing with residents in training in anesthesia, on whom we focus for the first several days or weeks or month in order to try to get them to master airway management, to master the use of an artificial airway, to establish appropriate conditions, and to successfully intubate. They don't do that very well under most circumstances during their month. They do gain a certain degree of proficiency, but when presented with a difficult case in the early stages of their residency, the chances are that they will get themselves into trouble and have to rely on individuals who are senior to them. The frightening part is that people are doing this every day as part of their living. The initial learning curve is pretty steep. I think it's one that has a number of pitfalls. Once it plateaus, it's impressive that they really struggle for long periods of time. A resident in a second or third year, even though he may have had a great deal of experience, will get into trouble and will have to be bailed out by someone who's far more experienced and, again, often without having to do anything much different. There are subtle little tricks.

I think you need to train physicians and others to intubate on an urgent basis, but I think that you have to emphasize that this is something that really should never be taken lightly. One really should not be misled into thinking that he is a master of the airway. In fact, I believe the anesthesiologist actually becomes less cavalier and more conservative as he gets older because he's been burned so many times, and he's been burned in circumstances in which he couldn't predict ahead of time that he was going to get into trouble. So, it's something to which a lot of thought needs to be given, in terms of who does it, under what circumstances, and what the resources are in terms of bailing them out if they fail or if they produce additional problems.

Durbin: I'd like to restate your first point about airway position in maximizing the opportunity for intubation, and I'd like to raise one other issue. It's my belief that more patients are harmed not by the failure to recognize a difficult airway, but by the failure to recognize esophageal intubation. This is supported by the closed-claim literature and a number of other places. Do you believe that end-tidal CO_2 monitoring is or should be state of the art and be required to establish tube location in all emergency and elective intubations?

Wilson: Yes I do. I think when one begins to look at much of the literature that has been published in the past several years, it is hard to argue that many of the techniques that we were taught to confirm position of a tube—visualization, auscultation of the chest, auscultation of the abdomen, humidity in the tube, pulse oximetry (which we know is limited in terms of its time response)—a lot of those in fairly well-done studies[1] in which individuals were blinded to procedures to assess esophageal airway intubation, the one technique that turns out to be the most reliable is the presence of CO_2 in the exhaled gas after the first 3 or 4 breaths. That's true especially when you've been ventilating an individual by mask. Not being an advocate of additional monitoring and, historically, being an advocate of using common sense and critical skills, I must admit that I have been convinced that CO_2 monitoring (certainly in the operating room where it is readily available under most circumstances, but I think, more importantly, with the portable disposable techniques that are available) should be used wherever possible to confirm the placement of the endotracheal tube. Again, the other 'confirmation' techniques are not reliable, and a number of closed claims support the fact that experienced individuals have sworn that the tube was in place, when, in fact, it wasn't, which then becomes a very costly event.

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Reines: Let me just throw in a little warning about some of the new CO_2 monitors that have become available, especially the monitors that turn purple when they have CO_2 in them. One problem we have found is that if the stomach is full of beer, there's enough CO_2 released from the beer that you can actually be misled by that. The second is that in a cardiac arrest in which you have very little circulation and no CO_2 is being produced, you may extubate thinking you're not in the trachea; so, there still is no sure way. My concern in setting a standard is that there are always going to be exceptions, and those are the ones that are going to hurt us.

Wilson: It's like any other standard. You know you can set a standard for EKG monitoring under a variety of circumstances, but that doesn't necessarily mean that it's going to be foolproof. Perhaps a better word is a guideline. CO_2 monitoring is certainly very useful; however, like any other technique it has its deficiencies (some related to the particular device and some to the physiology). But, again, when compared to other techniques in terms of reliability of assessment, it ranks right up there.

Hess: Could I just piggyback on the comments that have been made? I will talk about some of these things later when I talk about monitoring, but the usefulness of exhaled CO_2 to detect the proper placement of an endotracheal tube really depends a lot on whether the heart's beating.

Wilson: That's sort of academic, though, isn't it? I mean, if the heart's not beating that's obviously another important issue.

Hess: Yes, but it makes it difficult to use those devices to make sure that the tube's where you think it is. We reported some data at the AARC meeting this year:[1] We looked at 30 patients in cardiac arrest in the field. The Fenem End-Tidal CO_2 Detector was only useful to detect proper tube placement in 9 of the 30 patients.

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Wilson: Sure, I mean how reliable is the pulse oximeter if the heart's not beating. It's the same problem. It doesn't mean there's something wrong with pulse oximetry; it means that you have to relate the device that you're using to the physiologic circumstances.

Hess: Unfortunately, a lot of practitioners don't do that.

Wilson: Education.

Haponik: Would you please comment on the potential roles for CPAP and helium-oxygen mixtures as temporizing maneuvers. These can place pretty formidable demands on respiratory care practitioners.

Wilson: In the context of airway narrowing or airway pathology, I think heliox or use of helium has a role. The obvious limitations are the concentration of oxygen that the subject requires, which is obviously limited by the need to get the high concentrations of helium. This is an excellent temporizing maneuver, especially when one is dealing with suspected airway pathology in an individual who is otherwise stable prior to airway manipulation. You want to undertake noninvasive diagnostic maneuvers, such as tomograms and CTs, or other means to define the pathology as quickly as possible before undertaking specific interventions such as rigid or flexible endoscopy. So, as a temporizing measure, use of helium mixtures is certainly not questionable. I've had less experience with use of positive airway pressure. Individuals with high degrees of airway stenosis really don't respond very well to supplemental airway pressure. Usually at that time you can't do much to facilitate the exchange of gas with positive pressure, especially when the problem is a dynamic one in which exhalation is the issue. I talked only about stenotic lesions. In cases of tracheomalacia and bronchomalacia, especially where such lesions involve the lower airway, inspiration isn't as much a problem as is exhalation. I think assisting someone, again, with positive pressure by nasal route or oral route is okay, as long as you recognize the shortcomings and the potential for having that individual work harder, feel the stress, and actually develop more symptoms as he becomes more apprehensive.

Kacmarek: Let me ask the question you asked me, Roger. Assuming you were going to train a group of respiratory therapists to perform endotracheal intubation during CPR or on comatose patients, how would you design the program to ensure competency of those individuals?

Wilson: I think competency is a difficult question. I think the programs are fairly easy to design. The program should include a lot of training in anatomy and a lot of training in positioning. It's time-consuming in that trainees must go to the environment in which this is done electively, and that's, by and large, the operating room. They have to learn to use mannequins, which are a poor re-placement for the human population. Cadavers are okay, but obviously a bit distasteful to most individuals and often difficult to arrange in the hospital setting. Do it under controlled circumstances in the operating room, but make sure that you instill in all those being trained the notion that even though they have been successful once or twice they shouldn't be overconfident. This is something that if they need to do it in an urgent circumstance, they do it. I think that's the subtlety in it. I don't think you can truly certify an individual to do something that is as difficult and unpredictable as intubation when they're only doing it on a very low-frequency basis.

Emergency Management of Acute, Severe Asthma

Christopher H Fanta MD

Introduction

Case History

A 27-year-old malpractice lawyer with a long history of asthma presents to the Emergency Department with an exacerbation of her disease. Four days earlier she had developed a sore throat, "swollen glands," rhinitis, cough, and low-grade fever. Her husband had experienced a similar respiratory illness earlier in the week. Over the next 24-48 hours her symptoms seemed, in her words, to settle in the chest. She developed wheezing, chest tightness, congestion, and dyspnea on exertion. Her cough became productive of yellowish phlegm. She had spent most of the previous night seated in a reclining chair, too congested to sleep in her bed. Her usual asthma treatment consists of a slow-release theophylline preparation (300 mg twice daily) and an albuterol metered dose inhaler (2 puffs 4 times daily). However, during this illness she has used her albuterol inhaler as often as 6-8 times during the day and 2-3 times overnight. She decided to come to the emergency room tonight because of her shortness of breath and the impression that her inhaler was no longer working. She complains of a viselike grip around her chest.

On examination this young woman is seated tensely at the edge of the stretcher, her arms braced along the stretcher's edge. Her blood pressure is 150/90 mm Hg with an approximately 20-mm-Hg pulsus paradoxus (fall in systolic pressure with inspiration), heart rate 124/min and regular, respiratory rate 28 breaths/min with use of accessory muscles of respiration, and oral temperature 98.6°F. She is diaphoretic without cyanosis. Nares and pharynx are unremarkable. Her chest reveals bilateral inspiratory and expiratory wheezes in all lung fields. Because of her breathlessness, she refuses to lie supine for abdominal examination. She has no peripheral edema or calf tenderness. A subcutaneous injection of epinephrine is administered, and a blood sample is sent for immediate determination of theophylline level. A chest radiograph is ordered.

When she returns from the radiology department, she is quite breathless. The nurse requests that the physician re-evaluate the patient's condition. Chest auscultation reveals persistent wheezing, although perhaps less intense than earlier. Approximately 1 hour after receiving the sample, the chemistry laboratory reports the result of the serum theophylline concentration as 5.8 μg/mL (therapeutic range 10-20 μg/mL). The physician orders a second injection of epinephrine and intravenous administration of aminophylline. A reduced aminophylline loading dose of 3 mg/kg is given over 20 min, followed by a continuous infusion at $0.6 \text{ mg} \cdot \text{kg} \cdot \text{h}^{-1}$. Supplemental oxygen is given at 2 L/min by nasal cannula. The patient's room lights are dimmed, and she is advised to relax and to take slow, deep breaths.

Over the next several hours she gradually improves. Her diaphoresis, paradoxical pulse, and accessory muscle use resolve. Her chest now reveals only a few end-expiratory wheezes. She is able to walk approximately 100 feet to the bathroom and back without stopping to catch her breath. The emergency room physician decides that she is ready for discharge. He prescribes a 1-week course of oral antibiotics (ampicillin 250 mg 4 times daily) and advises her to increase her daily theophylline dose to 600 mg twice daily. She is

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Dr Fanta is the Clinical Director of the Respiratory and Critical Care Division at Brigham and Women's Hospital and Associate Professor of Medicine at Harvard Medical School—Boston, Massachusetts.

A version of this paper was presented by Dr Fanta on October 3, 1991, during the RESPIRATORY CARE Journal Conference on Emergency Respiratory Care held in Cancun, Mexico.

Reprints: Christopher H Fanta MD, Clinical Director, Respiratory and Critical Care Division, Brigham and Women's Hospital, 75 Francis St, Boston MA 02115.

told to contact her own physician first thing in the morning and to return to the Emergency Department should she again develop respiratory distress.

Changes in Management of Acute Asthma

Strategies for emergency department management of severe and life-threatening asthmatic attacks have undergone important changes over the past several years. These important changes have occurred despite the fact that no major new drugs have been introduced into our therapeutic armamentarium. Years ago the assessment and treatment received by the young lawyer presented above might have been considered consistent with the accepted standard of practice and then, as now, might have resulted in a successful outcome for the patient. However, based on current concepts of asthma management, her care was not optimal. It may have needlessly delayed her improvement and put her at risk for subsequent respiratory difficulties and an even more severe attack. With this case history as a point of departure, I review modern concepts of acute asthma care and discuss ongoing investigation and controversy. The recommendations set forth are consistent with the Guidelines for the Diagnosis and Management of Asthma[1] recently released by the Expert Panel convened by the National Institutes of Health as part of its National Asthma Education Program.

Bronchodilators and Bronchodilator Administration

Airway smooth-muscle contraction (bronchoconstriction) is an important cause of airflow obstruction in virtually all patients with acute exacerbations of asthma, and it is the cause most rapidly reversed with appropriate therapy. Consequently, the best way to improve lung function and alleviate respiratory distress in acute asthmatic attacks is by promptly and repeatedly administering bronchodilating drugs. Over the past 10-15 years, the numerous clinical trials conducted in emergency departments have helped to establish the relative potencies of various bronchodilators, the preferred route of administration, their potential for additive interactions, and their toxicities (both when given alone and in combination). A consistent finding of numerous studies is that sympathomimetics are the most potent available bronchodilating drugs for acute asthma.[2,3] When repeated doses are given in rapid succession (every 20-30 minutes), incremental bronchodilatation occurs following each successive dose.[4] Compared to intravenous aminophylline, repeated doses of sympathomimetics effect 3-4 times greater bronchodilatation over the first hour of emergency room care (Fig. 1).[2] The anticholinergic atropine sulfate is both less potent

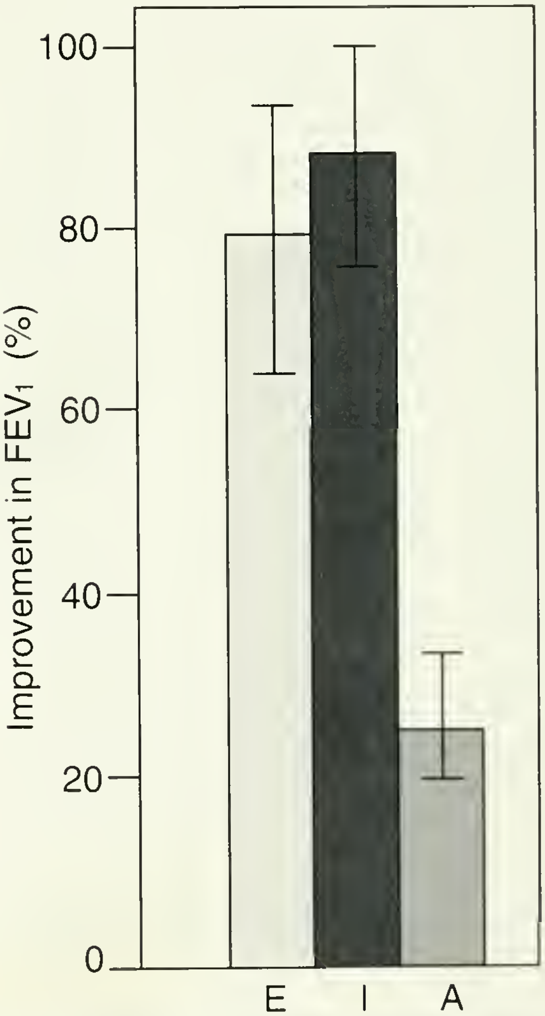

Fig. 1. Effects of epinephrine (E), isoproterenol (I), and aminophylline (A) in subjects with acute airway obstruction. The heights of the bars represent mean values, and the brackets represent 1 standard error. Responses to epinephrine and isoproterenol were both significantly greater than response to aminophylline.

and slower in onset when inhaled than are the inhaled sympathomimetics.[5] Even among patients who have been taking oral and/or inhaled sympathomimetics at the time that their asthmatic exacerbations develop, treatment with sympathomimetic bronchodilators in the emergency department remains the best approach to reversing airflow obstruction; clinically significant tolerance to their bronchodilator activity has not been observed.[6]

Parenteral versus Inhaled Bronchodilators

For many years it was assumed that parenteral administration of sympathomimetic bronchodilators was necessary for all but the mildest asthmatic attacks. It was hypothesized that severe airflow obstruction precluded adequate delivery of inhaled medication to peripheral airways; however, empiric observations have not supported this preconception. Randomized trials comparing subcutaneous epinephrine with inhaled beta-adrenergic agonists administered by continuous-flow small-volume nebulizer have found the inhaled agents to be at least as effective as the parenteral drug, even among patients with initial severe obstruction (forced expiratory volume in the first second, or FEV_1, ≤ 1 L or ≤ 35% of predicted).[2,7,8] Similar findings have resulted from comparisons between intravenous and inhaled beta agonists: When repeated doses are given in rapid succession, the inhaled route of administration produces bronchodilatation equal to that with continuous intravenous infusion.[9-12]

Theophylline Administration

In certain circumstances, concomitant use of bronchodilators from two different pharmacologic classes can produce additive bronchodilation in ambulatory patients with stable asthma.[13] For many years intravenous aminophylline was used in combination with sympathomimetic drugs for the treatment of acute severe asthma, with its use often reserved for the most severely obstructed patients in whom maximal bronchodilatation was sought. Extensive clinical evidence is now available to refute this approach; combining sympathomimetics and intravenous aminophylline produces no greater bronchodilatation than the repeated administration of sympathomimetics alone (Fig. 2).[3,7,14-16] Furthermore, adverse side effects are more frequent with the two-drug regimen. This observation pertains regardless of whether patients are taking theophylline at the time of their attack and independent of the severity of the attack. Immediate determination of the serum theophylline concentration followed by attempts to maximize the serum level as close as possible to the top of the therapeutic range by means of intravenous bolus and infusion of aminophylline is a misguided strategy for acute asthma management. In the emergency department setting, aminophylline is a relatively weak bronchodilator without additive effects beyond those achieved with inhaled beta agonists alone.

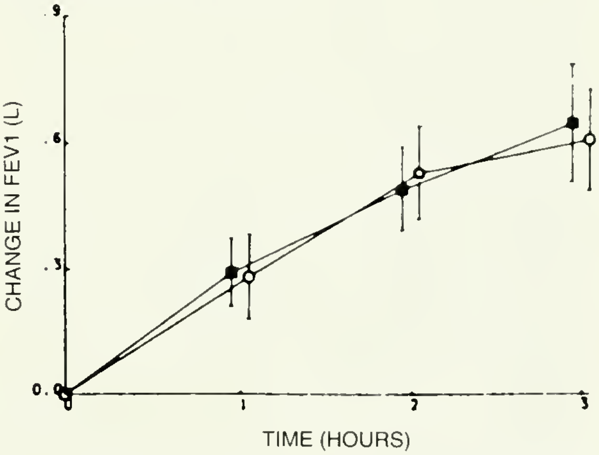

Fig. 2. Change in FEV_1 (mean ± SEM) in 20 patients treated with intravenous aminophylline and inhaled metaproterenol (O) and in 20 patients treated with inhaled metaproterenol alone (■). (Reprinted, with permission, from Reference 16.)

The study by Wrenn et al[17] found that when used in conjunction with inhaled beta-adrenergic agonists and corticosteroids, intravenous aminophylline reduced the frequency of hospitalization among asthmatic patients, although it did not bring about any greater improvement in lung function as measured by spirometry.[17] This curious finding needs to be interpreted with caution because it stands in contradistinction to a number of earlier studies[3,7,14-16] and cannot be explained by known actions of the drug. Unless future investigations can confirm the finding of Wrenn et al,[17] aminophylline is unnecessary in the emergency management of asthma.

Anticholinergic Agents

The potential for additive bronchodilatation when anticholinergic agents are used with beta-adrenergic agonists has also been explored with variable results. A small (10-20%) incremental benefit may be derived when ipratropium bromide solution (0.5 mg) is nebulized with conventional doses of beta-adrenergic agonists,[18,19] although not all studies have been able to demonstrate this additive effect.[20,21] It is possible that a subpopulation of asthmatic patients is uniquely responsive to anticholinergic stimulation, but such a subgroup has yet to be identified. Ipratropium bromide solution is not currently available for use in the United States, and other anticholinergic preparations (such as atropine sulfate[5] and glycopyrrolate[22]) have not been shown to be effective adjunctive therapy in the emergency treatment of asthma.

Sympathomimetic Side Effects and Doses

When multiple doses of sympathomimetic bronchodilators are given over a short period of time, unpleasant side effects are common: anxiety, tremulousness, palpitations, and headache.[16,23] On average, heart rate increases minimally and blood pressure tends to fall slightly.[2] Serious adverse consequences (such as significant cardiac arrhythmias or coronary ischemia) are rare,[24] especially when selective beta$_2$-adrenergic agonists are administered as aerosols. The safety of these medications given in relatively large doses has been best established in persons under the age of 50 who are free of complicating cardiovascular disease. The potential of these medications for significant toxicity in an older population or one with serious underlying heart disease deserves further investigation.

Medication Delivery

Years ago, continuous-flow nebulizers replaced intermittent positive-pressure breathing machines for medication nebulization in asthma; the continuous-flow nebulizers are simpler to set up, pose no risks of barotrauma from positive airway pressure, and can be used by patients with a minimum of supervision because only tidal breathing is needed for their use. However, medication delivery by simple continuous-flow nebulizer is inefficient because of droplet losses within the device itself, out the expiratory port, and in the oro- and hypopharynx. Thus, only a small percentage of the dose that is placed in the medication chamber actually reaches intrathoracic airways.[25] Recent trials[26,27] in emergency departments have explored the use of metered dose inhalers (MDIs) with spacer attachments as a simpler and less expensive method of delivery for inhaled beta agonists. For instance, in one study of asthmatic patients,[26] one group received metaproterenol administered by MDI (0.65 mg/puff) with a reservoir-bag spacer device (InspirEase) in a dose of 3 puffs (with an interval of 2 minutes between puffs) every 30 minutes; a second group of patients received aerosolized metaproterenol solution (15 mg) by continuous-flow nebulizer every 30 minutes. The investigators found no differences in improvement of lung function or relief of dyspnea between the two groups (Fig. 3). Despite data such as these, many centers

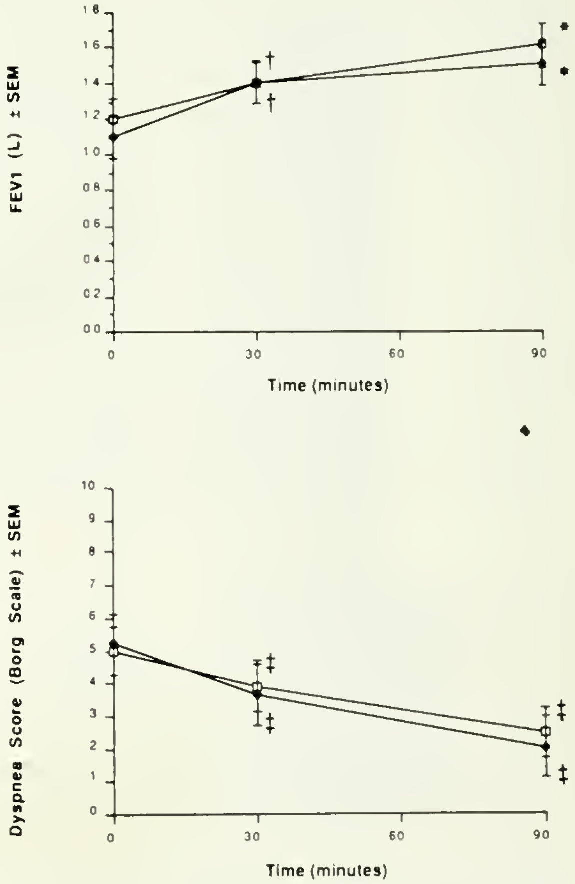

Fig. 3. Mean values for FEV$_1$ (± SEM) in upper panel and dyspnea scores in lower panel in 27 patients with asthma treated with metaproterenol via MDI and spacer (–□–) and in 26 patients treated with metaproterenol via nebulizer (--◆--). *p ≤ 0.001; †p < 0.05; ‡p = 0.005. (Reprinted, with permission, from Reference 26.)

remain reluctant to substitute MDIs with spacers for continuous-flow nebulizers for bronchodilator delivery in acute asthma because (1) although simple in concept, MDI use—even with spacer attachments—requires supervised administration because of the frequency of patient errors in delivery technique, making this a relatively labor-intensive method for health care providers; (2) dose-response studies in patients with chronic stable asthma indicate that much larger doses of beta agonists (up to 10 or more puffs from MDIs of standard formulation) are required to achieve maximal bronchodilatation,[28,29] the optimal dose to be given by MDI may vary from patient to patient and asthmatic episode to asthmatic episode and may be difficult to determine in advance; and (3) not all studies have found equivalent efficacy between the two delivery methods. In at least one study of newly hospitalized asthmatic patients with persistent severe obstruction, nebulized bronchodilator solution resulted in significantly greater improvement than did beta agonist by MDI.[30] It is possible that the larger administered dose of medication and simpler inhalational method accounted for the advantage of the continuous-flow nebulizers in this study.

This subject has been thoroughly reviewed in a special issue of RESPIRATORY CARE devoted to aerosol delivery (September 1991).[31] In particular, I refer you to Newman's article on aerosol generators and delivery systems.[32]

Care Delivery

The role of the respiratory therapist in administering inhaled medications to the acutely ill asthmatic patient varies among different institutions. In some emergency departments, respiratory therapists take direct responsibility for administration of each dose of inhaled medication; in others they provide training and backup for the emergency department staff (particularly nurses, who are continuously on hand in the emergency department for administering and monitoring treatments). In either case, an important general principle of care is that acutely ill asthmatic patients should receive prompt care: emergency services should be structured in such a way that treatment can begin within minutes of the arrival of severely ill patients. A common complaint voiced by patients with asthma relates to the prolonged delays that they have experienced prior to receiving emergency care.

Anti-Inflammatory Therapy

Mechanism of Bronchodilator-Refractory Asthma

Some patients with severe airflow obstruction fail to improve significantly despite intensive bronchodilator therapy with repeated doses of inhaled beta agonists. Although it would be easy to construe this failure to respond as being indicative of sympathomimetic resistance, or tachyphylaxis, switching to other classes of bronchodilator generally does not bring about reversal of the airways obstruction. In most instances, the correct explanation for a lack of improvement following aggressive bronchodilator therapy can be found in the pathophysiologic mechanism that underlies the airflow obstruction. In such patients, airway-wall inflammation and intraluminal secretions predominate over tracheobronchial smooth-muscle constriction. As a result, all medications that have as their primary action the relaxation of airway smooth muscle have only limited beneficial effect. When widespread mucus plugging obstructs airway lumina, and when edema and cellular infiltration narrow bronchi by increasing the thickness of bronchial walls, bronchodilating drugs can be expected to have minimal impact on lung function.

Role of Corticosteroids

Potent anti-inflammatory therapy is needed to treat the widespread mucus plugging, edema, and cellular infiltration of severe asthma; specifically, systemic corticosteroids are indicated. Without the use of systemic corticosteroids, some patients with severe asthma will have persistent severe airflow obstruction despite 24 or more hours of inhaled and parenteral bronchodilators; a small percentage will actually worsen despite bronchodilator treatment.[33] The addition of systemic corticosteroids speeds the resolution of severe attacks: A beneficial effect can be demonstrated within 12 hours of their administration;[33] however, the improvement observed with corticosteroid use is often very gradual. In one study of patients hospitalized with severe asthma, the average FEV_1 at the end of 24 hours of bronchodilator and steroid therapy was still only approximately 50% of normal. If one inspects the tracheobronchial tree of patients who have died from asthma, one finds intense eosinophilic bronchitis, widespread epithelial damage and sloughing, and extensive mucus plugging of airways leading to asphyxiation.[34] In view of these pathologic findings, it is not surprising that in severe, bronchodilator-refractory asthma, several hours to days are required for the clearing of tenacious secretions and cellular debris from the airways.

Given the delay in onset of action of systemic corticosteroids, their early institution in the emergency department is crucially important for patients who manifest inadequate improvement with bronchodilators alone. In general, such patients can be identified early in their course. Asthmatic patients who present with severe airflow obstruction and who, after 60-90 minutes of intensive bronchodilator therapy, continue to have an FEV_1 or peak expiratory flowrate (PEFR) < 40% of predicted should receive systemic corticosteroids. With infrequent exceptions, such patients will not experience significant further improvement over the ensuing 3 hours despite intensive treatment with inhaled and parenteral bronchodilators.[15] The need for systemic steroids can be established even earlier in some patients—those patients who develop severe asthmatic exacerbations despite regular use of oral corticosteroids and those patients with severe obstruction who actually deteriorate despite one or two treatments with nebulized bronchodilators. For some patients initiation or dose escalation of systemic corticosteroids will be inevitable, and earlier administration hastens the onset of beneficial effects.

Because of the delayed onset of action of corticosteroids and the slow tempo at which airway inflammation and mucus plugging resolve, one might predict that steroids would have a limited impact on the immediate outcome of acute asthmatic attacks. Indeed, three randomized trials conducted in emergency departments have all made the same observation:[35-37] Over the few hours that patients spend in the emergency department, the addition of an intravenous dose of corticosteroids at the onset of treatment does not significantly improve lung function beyond what is achieved with bronchodilators alone (Fig. 4). However, it is likely that in these trials some patients made adequate improvement with bronchodilators alone, and no further benefit could be detected with corticosteroids.[35] Other patients probably benefited from the anti-inflammatory effects of the steroids, but the benefit became manifest only many hours after these patients left the emergency department. Patients with exacerbations of asthma probably should not be detained in the emergency facility proper for more than 4-6 hours, and, thus, treatment with systemic corticosteroids should be begun with the intent of improving the course of the patient's response after he or she has left the emergency department.

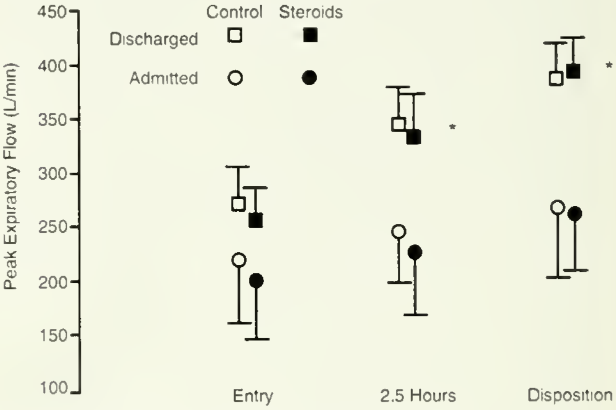

Fig. 4. Relation of peak expiratory flow (PEF) to duration of emergency room treatment. Means and error bars (95% CI, 1.96 SD) are shown. Entry time was approximately 0.5 hours after initiation of treatment. The second PEF determination was done approximately 2 hours later. Disposition PEF was measured at approximately 6 hours after arrival in discharged patients and 12 hours after arrival in admitted patients. At no time did the PEFs differ between the control and steroid groups. PEFs were similar on entry in the groups eventually admitted and eventually discharged. After 2.5 hours of therapy, PEF was significantly reduced in the group requiring hospital admission. $^*p = 0.002$, by analysis of variance. (Reprinted, with permission, from Reference 37.)

Along this same line of reasoning, an important role for corticosteroids in the management of acute asthmatic attacks is their use in the discharge regimen of patients being sent home after treatment in the emergency department. An asthmatic attack is rarely over at the time of discharge. Even though patients may report an absence of wheezing and have a clear chest on examination, at discharge they will almost uniformly have some degree of residual airflow obstruction.[38] This residual obstruction together with persistent lability of airway caliber (manifested as highly variable FEV_1 and PEFR) puts asthmatic patients at risk for recurrent severe attacks. Acute, life-threatening episodes not infrequently occur in the hours and days following treatment for acute asthma. The risk of developing a recurrent severe attack or relapse can be significantly reduced by a course of oral corticosteroids begun when the patient is discharged from the emergency department. Two randomized controlled trials[39,40] found strikingly similar results with the frequency of emergency department relapses reduced from approximately 22% to approximately 6% by an 8-day course of prednisone or methylprednisolone (Fig. 5). In addition, even among those whose relapse did not bring them back to the emergency department, fewer patients in the steroid-treated group reported disabling symptoms 7-10 days after discharge.[39]

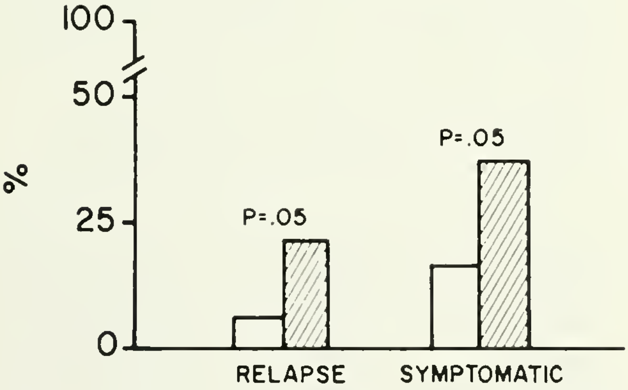

Fig. 5. Corticosteroid administration (☐) resulted in a decrease in relapse rate from approximately 22% to approximately 6%. ▨ = placebo. (Reprinted, with permission, from Reference 39.)

Patient Assessment

The care given to the malpractice lawyer (patient) described in our hypothetical case was inadequate in a number of ways. Despite the severity of her asthmatic attack, she received submaximal bronchodilator therapy. Sympathomimetic drugs were administered infrequently, and there was reliance on an increase in the serum theophylline concentration to treat persistent symptoms. Corticosteroids were not given in the emergency department and were not included in the post-discharge treatment plan. Instead, she was prescribed a course of antibiotics, despite the absence of clinical evidence in her case history for a bacterial bronchitis or sinusitis and the lack of scientific evidence indicating benefit from antibiotic therapy in acute asthmatic attacks. In general, antibiotics can be withheld unless fever, leukocytosis, sputum purulence (with polymorphonuclear leukocytes), pulmonary infiltrates on chest x-ray, or sinus tenderness with purulent nasal drainage point to a likely bacterial infection of the respiratory tract.

Objective Measures of Lung Function

A further shortcoming was the failure to make objective measurements of lung function (spirometry or PEFR determination) to assess the severity of her attack and its response to treatment. Subjective estimates by health care providers of the severity of airflow obstruction in asthma are notoriously inaccurate, with both over- and underestimates of the degree of obstruction being common (Fig. 6).[41] The failure of physicians to make adequately intensive interventions because of misjudging the severity of attacks is recognized as an important contributing factor to many deaths from asthma.[42] In the

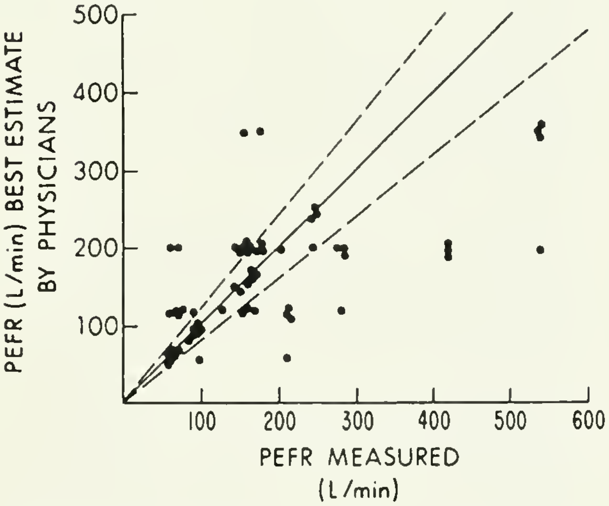

Fig. 6. Physicians' estimate of PEFR plotted against the measured value. The line of identity and 20% error margins are shown. Twenty seven of 62 estimates (44%) are within 20% of the measured value. (Reprinted, with permission, from Reference 41.)

case described, several clinical clues suggested that this was a particularly severe attack. A pulsus paradoxus ($\geq$ 12 mm Hg), use of accessory muscles of respiration, diaphoresis, and refusal to lie supine all indicate severe airflow obstruction and generally correlate with an $FEV_1 \leq 1.25$ L.[43,44] However, it is important to note that absence of these findings is not reliable evidence for mild obstruction because these physical signs may be absent in as many as half of asthmatic patients with severe obstruction.[45]

Inexpensive and easy-to-use devices for measuring expiratory airflow are now readily available, and repeated measurements of lung function should be a routine part of care for virtually all acutely ill asthmatic patients. Instead of qualitative estimates of severity based on clinical findings, one can rapidly obtain quantitative recordings and monitor change over time. At the present time, the proposed importance of basing therapeutic decisions in acute asthma care on objective measurements of lung function derives from expert opinion[1] rather than from clinical studies demonstrating improved outcomes. The potential utility of this approach is evident in three examples.

First, PEFR correlates inversely with partial pressure of arterial carbon dioxide (P_{aCO_2}) in acute asthma. In the absence of respiratory depressant medications, hypercapnia is rarely encountered unless the PEFR falls to $\leq$ 25% of normal.[46] In patients with severe asthmatic symptoms persisting despite initial bronchodilator therapy, peak flow determinations can be used to screen for patients at risk for ventilatory failure, and unnecessary arterial blood sampling can be avoided if the PEFR is > 25% of predicted.

Second, patients failing to improve significantly with intensive bronchodilator treatment can be identified early in their course with the use of objective measurements of lung function. As noted above, patients with an initial PEFR or $FEV_1 \leq 40\%$ of predicted who after 60-90 minutes of intensive treatment continue to have values $\leq$ 40% generally make little further improvement over the ensuing 3-4 hours despite continued inhaled and parenteral bronchodilators.[15] Thus, soon after their presentation, patients can be identified who (1) are likely to benefit from systemic steroid therapy and (2) are at increased risk of requiring hospitalization for their asthma.

Third, measurement of lung function can help assess a patient's risk for recurrent severe disease following emergency department discharge and so can affect the decision regarding need for hospitalization. In a study of patients whose discharge from the emergency department was based solely on traditional clinical criteria (relief of dyspnea and absence of wheezing), the relapse rate was significantly greater among patients with lower discharge values for FEV_1.[45] In a number of investigations summarized elsewhere,[47] it was found that patients successfully discharged from several different emergency departments following treatment for asthmatic exacerbations had an average PEFR of 70-75% of predicted, whereas patients who subsequently developed recurrent respiratory difficulties on average had values of 50-55% of predicted at the time of emergency department discharge. For patients requiring hospitalization, the average PEFR at the time of hospital admission was 35-45%.[47] Data such as these led the Expert Panel of the National Asthma Education Program to incorporate lung function measurements in their recommended criteria for deciding which asthmatic patients should be hospitalized for care.[1]

Therapists as Evaluators and Instructors

Respiratory therapists are commonly called upon to test the pulmonary function of asthmatic patients acutely ill with cough and dyspnea. In all but the most desperately ill patients (those too breathless to speak), FEV_1 or PEFR can be measured without risk to the patient. Supportive but forceful coaching of the patient is needed to enlist patient cooperation and ensure maximal expiratory effort. The importance of accuracy in performing the tests and recording the results cannot be overemphasized because erroneous data may affect management and jeopardize patient care.

Respiratory therapists who have contact with patients close to the time of their discharge home have a special opportunity to provide meaningful instruction. In some patients, this teaching might take the form of instruction in the use of home peak flowmeters: in almost all patients, it should include reinforcement of the proper technique for using metered dose inhalers—with or without spacers. Patients recovering from an often frightening episode of their asthma frequently will have many questions about their disease and its management. By devoting a few minutes to patient teaching in the emergency department, a well-informed respiratory therapist can have a major impact on outpatient care and may help to prevent a subsequent life-threatening attack.

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Fanta Discussion

Kacmarek: Just a general comment about some of the data that you presented regarding MDIs and small volume nebulizers. In all the studies that I reviewed in which results were not equivalent, the lack of equivalence was associated with the patient's inability to master the MDI technique.[1] If a spacer was added, then there was equivalence. I am trying to remember the study that you showed, in which a difference was present that could not be traced to improper technique or nonuse of a spacer.

1. Cushley MJ, Lewis RA, Tattersfield AE. Comparison of three techniques of inhalation on the airway response to terbutaline. Thorax 1983;38:908-913.

Fanta: Your point is well taken. The use of the MDI with or without a spacer requires a lot of coaching for proper delivery, especially in acutely ill, dyspneic patients. Spacers are certainly an important inhalation aid in the acutely ill patient who has trouble coordinating metered dose inhalers, but I think that their use also requires supervision for proper administration. The study mentioned in my talk, which found greater bronchodilatation with continuous-flow nebulizers than with MDIs,[1] did use an inhalational aid (InspirEase reservoir bag); it is not mentioned in that report whether medication administration was directly supervised by a therapist or other hospital staff.

1. Morley TF, Marozsan E, Zappasodi SJ, Gordon R, Griesback R, Giudice JC. Comparison of beta-adrenergic agents delivered by nebulizer vs metered dose inhaler with InspirEase in hospitalized asthmatic patients. Chest 1988;94:1205-1210.

Kacmarek: I agree with you 100%. I'm not advocating MDIs in the setting of an acute asthmatic attack. Patients who are that stressed do better with the small volume nebulizer. However, in most other patients the dosing and the accuracy with which you can predict the quantity of drug received is better using an MDI with a spacer.

Stoller: It seems that aminophylline, despite all of the information you presented, dies a slow death, and is being resurrected by Wrenn and his colleagues in the *Annals of Internal Medicine* in August 1991.[1] I wonder what your thoughts and reactions are to this kind of "mini resurrection."

1. Wrenn K, Slovis CM, Murphy F, Greenberg RS. Aminophylline therapy for acute bronchospastic disease in the emergency room. Ann Intern Med 1991;115:241-247.

Fanta: In the study that Jamie (Stoller) refers to, patients presenting to the emergency room were randomly assigned to receive nebulized bronchodilators, intravenous corticosteroids, and intravenous aminophylline versus nebulized bronchodilators, intravenous corticosteroids, and placebo. Their response was measured by spirometry at the end of 2 hours of treatment. There was absolutely no difference between the aminophylline- and placebo-treated groups in terms of lung function at the end of 2 hours; however, it turned out that the aminophylline-treated group had a reduced need for hospitalization. It is unclear to me how to explain this finding when the levels of lung function in the two groups were identical. One can either believe in alternative, unmeasured effects of aminophylline or one can say that this study stands as a single and unique observation that is different from all the previous observations and my own guess would be different from subsequent trials that will try to repeat their findings. I'd be interested to hear what you have to say, Jamie.

Stoller: I agree. Actually, Regis McFadden wrote a thoughtful accompanying editorial[1] regarding the criteria for discharge, which, as I recall the report, putatively were based on some pre-established guidelines in the emergency room for discharge: but my methodologic question about that paper is, How strictly were the criteria being applied in both groups, even though in theory the house officers were blinded. One can't exclude the possibility of a bias related to patients' reporting subjective tremulousness associated with the aminophylline, those sorts of subtle unblinding events that can occur in such a study, as you all know.

My second question regards management of the intubated asthmatic. The specific question regards PEEP. The original letter to the editor by Qvist and his colleagues[2] talks about PEEP as a pneumatic stent, the possibility of auto-PEEP, and the intriguing possibility that PEEP is actually facilitating ventilation and facilitating ultimate weaning by decreasing work of breathing by opposing the imposed inspiratory thresholds of auto-PEEP. I wonder what your own experience and feelings are regarding PEEP as an adjunct to the pharmacologic management of the ventilated asthmatic patient.

1. McFadden ER Jr. Methylxanthines in the treatment of asthma: the rise, the fall, and the possible rise again (editorial/comment). Ann Intern Med 1991;115:323-324.
2. Qvist J, Andersen JB, Pemberton M, Bennike KA. High-level PEEP in severe asthma (letter). N Engl J Med 1982;307:1347-1348.

Fanta: Quite frankly, Jamie, I think that is beyond my area of expertise, and I suspect that there are people in the audience much better able to answer that question than I. I think that I understand the theoretical benefit from the use of PEEP in this setting, to relieve some of the work of the inspiratory muscles, which are, curiously, active during the expiratory phase of respiration in patients with severe obstructive lung disease. But whether that can be translated into a clinical benefit, I cannot comment based on my personal experience.

Weaver: For severe asthmatics we commonly allow hypercapnia, as you allude. Could you comment on other therapies for severe asthma, refractory asthma, specifically the use of magnesium, ketamine, paralysis, those sort of modalities.

Fanta: Unfortunately, for most of these 'heroic measures' used to treat life-threatening asthma, I think there is only anecdotal supportive evidence. There has been one controlled trial[1] of intravenous magnesium in which patients who had been treated with beta-agonist bronchodilators but still had severe airflow obstruction (average peak expiratory flow < 250 L/min) were randomized to intravenous magnesium at a dose of 1.2 g, given over 20 minutes, or to a placebo infusion. The placebo-treated patients had a stable course over the ensuing 45 minutes. The magnesium-treated patients had further bronchodilatation that was statistically significant at 20 to 45 minutes (Fig. 1). This one study suggests that in this setting, magnesium sulfate is in fact an effective bronchodilator. Whether it is more effective than other strategies, I think is yet to be determined, and there's lots to be learned about its use, including the optimal dose, how often to give it, and what its toxicities are. At the present time, this remains preliminary work. Other therapies (such as inhalational anesthetic agents, intravenous ketamine, bronchoalveolar lavage, and whole-lung lavage, with or without acetylcysteine, intravenous isoprotere-nol, and the like) have been described only for a very limited number of patients, either case reports or small series.[2,3] My own experience suggests that in most instances—certainly not all, but in most instances—the best strategy is to stick with conventional methods and wait. Even very severe asthmatic exacerbations generally improve with time and anti-inflammatory therapy with corticosteroids. If the patient can be supported over that interval, one can

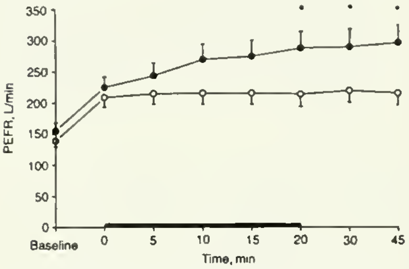

Fig. 1. Mean peak expiratory flowrates with standard errors. Placebo-treated = O; magnesium sulfate-treated = ●.

avoid some of the complications associated with these other heroic measures.

1. Skobeloff EM, Spivey WH, McNamara RM, Greenspon L. Intravenous magnesium sulfate for the treatment of acute asthma in the emergency department. JAMA 1989;262:1210; 1213.
2. Noppen M, Vanmaele L, Impens N, Schandevyl W. Bronchodilating effect of intravenous magnesium sulfate in acute severe bronchial asthma. Chest 1990;97:373-376.
3. Okayama H, Okayama M, Aikawa T, Sasaki M, Takishima T. Treatment of status asthmaticus with intravenous magnesium sulfate. J Asthma 1991; 28:11-17.

Porter: Could you comment on your experience with the problem you addressed near the end of your lecture at > age 45, where ischemic or nonischemic left-ventricular dysfunction may be responsible for the wheezing that's heard in the emergency department, and whether you've seen benefits, no effect, or detriment with beta-agonist-inhaled therapy in that setting?

Fanta: The practice in our hospital has been to use inhaled selective beta$_2$-adrenergic agonists as first-line therapy even in the older population. Even in patients with known coronary artery disease and a history of coronary ischemia, beta agonists remain beneficial in terms of their bronchodilator activity and reduction of stress associated with respiratory distress. It is worth emphasizing, however, that our knowledge of the relative toxicities of the different bronchodilating agents—comparing inhaled beta agonists to aminophylline, or to the anticholinergics—is extremely limited, especially in an older, 'at risk' population. In practice, we sort of muddle through, often reducing by half the dose of the inhaled beta agonists and hoping for a minimum of ischemic or arrhythmic complications.

Thompson: Do you have a criterion for intubation? At what point do you intubate an asthmatic?

Fanta: I find that a very difficult question. If I were to suggest any one value for the P_{aCO_2} at which patients with asthma were to be intubated, we would all find reasons to disagree with it. Probably most important is the *trend* in P_{aCO_2}. In general, the patient with a $P_{aCO_2} \geq 50$ torr, who appears not to be improving or is worsening despite maximal therapy, is the patient who should be intubated and mechanically ventilated. Obviously, patients who are apneic or obtunded and no longer able to cooperate with inhaled medications also should be intubated. In most instances, though, I think that it's very much still a judgment call—which patients you think may improve with a little more time and a little more therapy, and which patients clearly are worsening in spite of your therapy and should be intubated. Again, I think there are a lot of experts in the audience who might have comments that differ from mine.

Kacmarek: Chris, I want to ask you about dosing during an acute asthmatic attack. The 'standard' dose of bronchodilator is frequently the same dose that's used, at least initially, in the emergency room when a patient presents in an acute asthmatic attack. What is your philosophy on bronchodilator dosing in this setting?

Fanta: I think that's a wonderful question because it gets at another unknown: What is the optimal dose of inhaled beta-agonist bronchodilator in acutely asthmatic patients? We've used 7.5 mg of albuterol by continuous-flow nebulizer every 20 minutes for 3 doses. At 7.5 mg over 1 hour, it seems like a pretty big dose, but whether it's optimal and whether more could safely be given without significantly greater side effects are unknown. It should be addressed in emergency department studies; we should find out.

Hess: Along those same lines, what are your feelings about continuously aerosolized bronchodilator? There have been some anecdotal reports,[1] but as far as results from controlled clinical trials are concerned, I haven't been convinced that it makes any difference. I wonder what your feelings are.

1. Moler FW, Hurwitz ME, Custer JR. Improvement in clinical asthma score and P_{aCO_2} in children with severe asthma treated with continuously nebulized terbutaline. J Allergy Clin Immunol 1988;81:1101-1109.

Fanta: I have a colleague who when asked how much beta agonist should be given in severe asthma replies one dose per breath. Continuous nebulization is one way to achieve that goal. However, in at least one reported trial,[1] no difference in bronchodilatation could be found between continuous nebulization and intermittent dosing (for instance, nebulization every 20 minutes). I must say that it takes about 10 to 12 minutes to evacuate one of those small volume nebulizers, so that every 20-minute dosing leaves relatively little 'down time.' Again, this question gets us back to the issue of optimal dose. How much and how often should we be using our beta agonists, especially if we're focusing on these drugs as our prime bronchodilator therapy in the emergency room? We need to know more about dosing and how best to deliver them.

1. Colacone A, Wolkove N, Stern E, Afilalo M, Rosenthal TM, Kreisman H. Continuous nebulization of albuterol (salbutamol) in acute asthma. Chest 1990;97:693-697.

Mathews: I would just like to comment for a moment on the preventive aspect of these acute life-threatening asthmas. Nelson, Saks, & O'Connell, in *Mayo Clinic Proceedings* back in November of '89, published an excellent article on life-threatening asthmas and status asthmaticus in children.[1] They suggested that perhaps the best cure is prevention, individualized maintenance, and education programs for the patient. I was wondering if you see this as a role for the respiratory therapist, such as in Better Breathing Clubs, or the chronic lung clubs, and the children's asthma clubs?

1. Nelson DR, Saks MI, O'Connell EJ. Approaches to acute asthma and status asthmaticus in children. Mayo Clin Proc 1989;64(11):1392-1402.

Fanta: That's an excellent comment, and I thank you for making it. As your comments suggest, the emergency room is a very appropriate place to institute preventive strategies for severe asthmatics. The respiratory therapist has a perfect opportunity to make those interventions, being a health care provider knowledgeable about asthma care. Having just come through a frightening, perhaps life-threatening event, the patient is likely to be very receptive to any ideas that you can share about preventing recurrences. Let me specifically mention two areas in which respiratory therapists could be very helpful. (1) More and more patients will be sent home with peak flowmeters to monitor their lung function and to alert them as to when they're getting into trouble and should contact their physician or return to the emergency room. It would be very useful for respiratory therapists who are familiar with this equipment to instruct patients on its use and the recording of their peak flow measurements at home. (2) The second area is instruction in the proper use of metered dose inhalers. Even the most experienced asthmatic patients make mistakes in their use of these devices; reviewing their correct use would be a perfect teaching opportunity for respiratory therapists.

Diagnosis and Management of Massive Hemoptysis: A Review

James K Stoller MD

Introduction

Hemoptysis, the coughing of blood, is a common clinical symptom, responsible for 6.8% of outpatient chest clinic visits,[1] 11% of admissions to a hospital chest service,[2] and 38% of patients referred to a chest surgical practice.[3] In addition to the diagnostic challenge posed by the patient with hemoptysis, the subset of patients who cough large amounts of blood (ie, those with massive hemoptysis) poses additional challenges in acute and long-term management. In this paper, I review the diagnosis and management of massive hemoptysis and after defining massive hemoptysis and reviewing the underlying pulmonary vascular anatomy, consider the differential diagnosis and epidemiology of massive hemoptysis. Because it is essential for understanding management, the natural history of massive hemoptysis is reviewed next, and then attention is turned to diagnostic modalities, including chest imaging, bronchoscopic techniques, and nuclear medicine modalities. Finally, treatment strategies are considered, including local airway treatments (ie, bronchial tamponade, lavage, topical therapy), bronchial and pulmonary angiography, and intubation strategies. The efficacy of surgical resection is considered, followed by a critical review of literature regarding medical versus surgical therapy of massive hemoptysis.

Definition of Massive Hemoptysis

As summarized in Table 1,[4-9] different definitions of hemoptysis have been presented, reflecting a range of views about how much blood loss comprises "massive" hemoptysis and alternate views that the clinical consequences of hemoptysis matter more than the actual volume of blood coughed. Clearly, to the extent that the volume and sequelae of expectorated blood are important determinants of patient outcome,[6,7,10] varying definitions of massive hemoptysis among series complicate the task of comparing outcomes and management recommendations. The cautious reader will take account of this, and in this review I attempt to specify the definition of massive hemoptysis as needed.

Table 1. Definitions of Hemoptysis

- Massive hemoptysis
  - Spectrum of "volume" definitions
    - ≥ 100 mL/24 h[4,5]
    - > 600 mL/48 h[6]
  - Major (≥ 200 mL/24 h) vs massive (≥ 1,000 mL/24 h)[7]
  - "Magnitude of effect" definitions[8]
    - Presents risk of large aspiration
    - Life-threatening by virtue of airway obstruction, hypotension, or anemia
- Exsanguinating hemoptysis[9]
  - Hemoptysis that threatens because of actual volume of blood lost
  - Loss of ≥ 1,000 mL total at rate of ≥ 150 mL/h

Dr Stoller is Head, Section of Respiratory Therapy, and Staff, Department of Pulmonary Disease, Cleveland Clinic Foundation, Cleveland, Ohio.

A version of this paper was presented by Dr Stoller on October 3, 1991, during the RESPIRATORY CARE Journal Conference on Emergency Respiratory Care held in Cancun, Mexico.

Reprints: James K Stoller MD, Department of Pulmonary and Critical Care Medicine, Cleveland Clinic Foundation, 9500 Euclid Ave, Cleveland OH 44195-5038.

The most lenient volume definition of massive hemoptysis has been proposed by Amirana et al[4] and Bobrowitz et al:[5] expectorating ≥ 100 mL of blood a day at least once. At the other extreme of volume definitions are those proposed by Crocco et al[6] (> 600 mL of expectorated blood in 48 h) and Corey and Hla[7] (≥ 1,000 mL of expectorated blood/24 h).

As an alternate to volume definitions of massive hemoptysis, other authors have defined massive hemoptysis by the magnitude of the clinical consequences. For example, based on the fact that the anatomic dead space of the major airways is 100-200 mL in most individuals, massive hemoptysis has been defined as the volume of expectorated blood that presents risk for large aspiration or that is life-threatening by virtue of airway obstruction, hypotension, or blood loss. As specific examples, Holsclaw et al[8] offer three criteria for massive hemoptysis: pulmonary bleeding that (1) causes death or requires hospitalization, (2) is large enough to give laboratory or clinical evidence of systemic blood loss, or (3) requires blood or plasma transfusion.

Garzon et al[9] distinguish "exsanguinating" hemoptysis as pulmonary bleeding that is brisk enough to threaten life not only by asphyxiation but also by blood loss itself; a volume of ≥ 1,000 mL at a rate of ≥ 150 mL/hour is proposed.

Amidst these extremes of volume and magnitude-of-effect definitions, other distinctions have been made in defining massive hemoptysis. For example, Corey and Hla[7] distinguish between major hemoptysis (volume ≥ 200 mL/24 h, but < 1,000 mL/24 h) and massive hemoptysis (volume ≥ 1,000 mL/24 h). However, the practical difficulty of measuring the volume of expectorated blood in usual clinical circumstances limits the value of finer distinctions in defining massive hemoptysis. For purposes of this review, studies encompassing the whole spectrum of massive hemoptysis definitions are considered, but specific definitions will be provided as needed for clarity of interpretation.

Vascular Anatomy of the Lung

Sources of pulmonary bleeding include the pulmonary circulation, a low-pressure circuit (with normal pulmonary artery pressures of 15-20 mm Hg systolic and 5-10 mm Hg diastolic), and the bronchial circulation, consisting of bronchial arteries (which branch from the aorta and have systemic arterial pressures), and bronchial veins (which drain into the systemic veins to the right heart). As depicted in Figure 1, normal bronchial and pulmonary circulations are connected such that bronchial arterial blood feeding the proximal airways (eg, trachea, main-stem bronchi) drains into bronchial veins and empties into the right heart, whereas bronchial arterial blood feeding the intrapulmonary airways and lung tissue drains through bronchopulmonary anastomoses into the pulmonary veins and left heart.

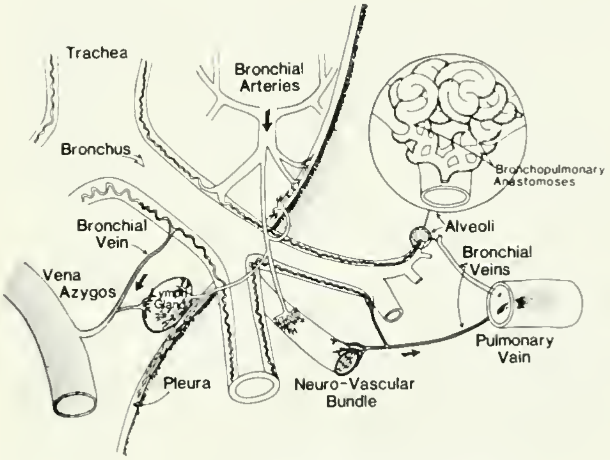

Fig. 1. Schematic of the systemic blood supply to the lung. Note that the flow from the extrapulmonary airways and supporting structures returns to the right heart, whereas intrapulmonary flow becomes anastomotic with the pulmonary circulation and returns to the left heart. (Reprinted, with permission, from Reference 10.)

Bronchial arteries arise either directly or indirectly from the aorta, at the level of the third through the eighth thoracic vertebra (T3-T8), most frequently at T5-T6.[11] Patterns of bronchial arterial branching from the aorta have been examined in both human postmortem studies[12] and during planned embolization therapy for massive hemoptysis.[10,13] For example, among 72 patients undergoing bronchial angiography, Uflacker et al[13] observed 10 different patterns of bronchial arterial anatomy, 5 of which accounted for 87.4% of observations. The three most common patterns were (1) a single intercostal trunk on the right from which the right bronchial artery arises and a single left bronchial artery (30.5%); (2) an intercostal trunk on the right giving rise to the right bronchial artery and a second common trunk from which right and left bronchial arteries arise (25.0%); and (3) a right intercostal trunk giving rise to a bronchial artery and 2 bronchial arteries on the left (12.5%). Although the anterior spinal artery of Adamkiewicz has been reported to arise from the bronchial artery in up to 5% of patients[12] (precluding embolization because of the risk of spinal artery infarction), no such instance was demonstrated in the series by Uflacker et al.[13]

Anastomoses between the bronchial arteries and tributaries of the pulmonary veins have been demonstrated in normal lungs at postmortem.[10] These communications permit drainage of bronchial arterial blood into the left heart, the volume of which may increase in chronic inflammatory conditions (eg, tuberculosis, bronchiectasis, cystic fibrosis). New collaterals from the bronchial arteries or adjacent intrathoracic systemic arteries (eg, phrenic, intercostal, subclavian, or axillary) to the affected lung parenchyma may also develop.[14] As I will discuss later, luxuriant collaterals from unexpected intrathoracic arteries can undermine the success of bronchial arterial embolization for massive hemoptysis and require more extensive arterial catheterization for control of bleeding.[10,14]

Differential Diagnosis of Hemoptysis and Massive Hemoptysis

The differential diagnosis of hemoptysis is presented in Table 2 and has been discussed amply in available reviews.[15-17] Changes in rates of disease occurrence (eg, declining rates of new-onset bronchiectasis) and diagnostic advances (eg, the advent of flexible fiberoptic bronchoscopy in the 1970s)

Table 2. Differential Diagnosis of Hemoptysis

- Infectious (bacterial, mycobacterial, viral, fungal, parasitic)
  - Pneumonia
  - Lung abscess
  - Bronchitis, acute or chronic
  - Bronchiectasis (including cystic fibrosis)
  - Mycetoma (eg, aspergilloma)
- Neoplasm
  - Bronchogenic cancer
  - Pulmonary metastasis from extrapulmonary primary
  - Other (eg, sarcoma, bronchial adenoma)
- Foreign body/trauma
  - Aspirated foreign body
  - Broncholith
  - Tracheovascular fistula (eg, tracheo innominate artery)
  - Trauma
  - Goodpasture's syndrome
  - Idiopathic pulmonary hemosiderosis
  - Other pulmonary vasculitis (eg, Bechet's syndrome, rheumatoid arthritis, systemic lupus erythematosus)
- Cardiac/pulmonary vascular
  - Left ventricular failure
  - Mitral stenosis
  - Pulmonary embolus/infarction
  - Pulmonary artery perforation (Swan-Ganz catheter mishap)
- Miscellaneous
  - Arteriovenous malformation (including Osler-Weber-Rendu syndrome)
  - Bronchial telangectasia
  - Catamenial hemoptysis
  - 'Pseudohemoptysis' with *Serratia marcescens* pneumonia
  - Pneumoconiosis
  - Indeterminate (2-15%)

Table 3. Estimates of Prevalence of Massive Hemoptysis

Author (Year)	Definition of MH	Patients with MH (n)*	Prevalence
Pursel & Lindskog,[2] 1961	> 500 mL single or > 1,000 mL/several days	5	5/105 (4.8%) patients with hemoptysis for thoracic surgical admission
Crocco et al,[6] 1968	> 600 mL/48 h	67	67/4,331 consecutive admissions (1.5%)
Garzon & Gourin,[18] 1978	> 600 mL/24 h	75	75/18,000 admissions to chest service (0.42%)
Corey & Hla,[7] 1987	≥ 1,000 mL/24 h	59	59/887 consecutive patients with hemoptysis (6.7%)
Rabkin et al,[19] 1987	≥ 500 mL/24 h	120	120/306 patients with hemoptysis for embolization (39%)

*MH = massive hemoptysis

Table 4. Causes of Massive Hemoptysis in Selected Series

Author (Year)	Definition	Patient Total	Cause of Massive Hemoptysis, % (n)									
			TB, Active	TB, Inactive	Lung Cancer	Bronchiectasis	Bronchitis	Abscess	Mitral	Fungoma	Unknown	Other
Crocco et al[6] (1968)	> 600 mL/48 h	67	49%(33)	24%(16)	8%(5)	10%(7)	—	9%(6)	—	—	—	—
Garzon & Gourin[20] (1974)	≥ 600 mL/24 h	62	26%(16)	47%(29)	3.2%(2)	11.3%(7)	—	6.5%(4)	—	—	—	6.5%(4)
McCollum et al[21] (1975)	Asphyxia threat	15	33%(5)		6.7%(1)*	20%(3)	—	—	—	13.3%(2)	6.7%(1)	20%(3)
Yang & Berger[22] (1978)	> 200 mL/24 h	20	25%(5)	25%(5)	15%(3)	15%(3)	—	—	5%(1)	5%(1)	10%(2)	—
Garzon et al[9] (1982)	> 1,000 mL/6 h	24	29%(7)	17%(4)	13%(3)	17%(4)	—	8%(2)	—	—	8%(2)	8%(2)
Uflacker et al[13] (1985)	NS†	75	76%(57)		—	1.3%(1)	—	2.6%(2)	—	16%(12)	—	3.9%(3)
Conlan et al[23] (1983)	NS	123	38%(47)	—	4.9%(6)	30%(37)	—	4.9%(6)	—	3.3%(4)	—	18.7%(23)

*Metastatic.

†NS = not specified.

likely account for a changing spectrum of causes of hemoptysis. In recent series, for example, chronic bronchitis occurs more commonly, and tuberculosis and bronchiectasis occur less commonly, than in earlier series.[7]

Available series (Table 3[2,6,7,18,19]) show that massive hemoptysis accounts for a minority of patients with hemoptysis (4.8-6.7% of hospitalized patients with hemoptysis,[2,7]) and that the spectrum of causes of massive hemoptysis is narrower, especially chronic bronchitis, bronchiectasis, tuberculosis (both active and inactive), bronchogenic cancer, lung abscess, and mycetoma (Table 4[6,9,13,20-23]). Also, unlike hemoptysis in general (for which tuberculosis and bronchiectasis have become more infrequent causes[7]), the spectrum of causes of massive hemoptysis has changed little among series that span three decades (Table 4). In addition to tuberculosis and bronchiectasis, lung abscess, lung cancer, and mycetoma have been consistent causes of massive hemoptysis over time.

Natural History of Massive Hemoptysis

To the extent that treatment benefits massive hemoptysis, descriptions of the natural history and prognosis of massive hemoptysis reflect the impact of therapies used. However, despite available treatment, massive hemoptysis may initially present explosively, resulting in acute cardiopulmonary embarrassment and death. For example, 7% of the 113 patients reported by Bobrowitz et al[5] presented with initial episodes of massive hemoptysis resulting in death within minutes of onset. In other series, estimates of the frequency of acutely fatal episodes of massive hemoptysis are 18.6% of 43 tuberculosis patients with massive hemoptysis,[24] 32% of 19 patients with massive hemoptysis from cystic fibrosis,[8] and 9% (2/22) and 10% (2/20) in two mixed series of patients.[24,25]

Notwithstanding this important minority of acutely fatal episodes, bleeding in most patients with massive hemoptysis can be successfully managed until the bleeding stops. For example, in a series of 33 tuberculosis patients managed medically and surviving the acute episode of massive hemoptysis (defined as ≥ 200 mL/24 h),[24] bleeding stopped in all patients within 5 days. Table 5 presents the course of bleeding in 64 medically

Table 5. Resolution of Hemoptysis in 64 Patients Successfully Managed Medically*

Resolution by	Cumulative %
Day 1 in 31.2%	31.2%
Day 2 in 17.1%	48.3%
Day 3 in 26.5%	74.8%
Day 4 in 12.5%	87.3%
Day 5 in 4.6%	91.9%
Day 6 in 3.1%	95.0%

*Adapted from Reference 5.

treated patients with massive hemoptysis (≥ 100 mL/24 h).[5] Bleeding stopped by Day 3 in approximately three quarters of patients and in virtually all patients by Day 6.

Once an acute episode of massive hemoptysis has resolved, management is dictated by the risk of recurrent massive hemoptysis. Indeed, patients with recurrent massive hemoptysis comprise many available series. For example, among 59 patients with massive hemoptysis (≥ 1,000 mL/24 h) reported by Corey and Hla,[7] 40% (24/59 patients) had bled previously. Among 67 patients bleeding > 600 mL/48 hours reported by Crocco et al,[6] 87% (58/67) had bled previously, 9 of whom had 4 or more previous bleeding episodes. Few of the earlier episodes were said to be massive, however.

Longitudinal estimates of recurrence risk for massive hemoptysis are scarce; however, in one available study, up to 20-year follow-up of a cohort of 728 cystic fibrosis patients showed that 45% of patients with massive hemoptysis experienced recurrence after conservative medical management.[26] On the other hand, overall survival of patients with massive hemoptysis in this series did not differ from survival in the group without massive hemoptysis. Other sources of estimates of recurrence risk after massive hemoptysis are series that report long-term follow-up after bronchial embolization therapy, which are reviewed in Table 6.[12,19,27-31] Overall, recurrence rates over long-term follow-up are 12% to 46% after bronchial embolization. Though these are not 'natural history' studies because recurrence rates reflect the impact of treatment, these follow-up rates can be considered to provide 'best case' rates. In the largest available series, Rabkin et al[19] report recurrent hemoptysis in 22.8% of 158 patients followed for 3 to 5 years after successful bronchial embolization. Uflacker et al[13] report rebleeding in 21% of 56 patients for

Table 6. Success Rates of Bronchial Artery Embolization for Massive Hemoptysis

Author (Year)	Patients (n)	Cause of MH*	Success Rate (MH Arrested)	Comment
Wholey et al,[27] 1976	5	Mixed	100% (immediate)	First report
Remy et al,[28] 1977	49	Mixed	84% (immediate); 71% (long-term)	———
Fellows et al,[29] 1979	13	CF	93% (short-term); 54% (long-term)	13% unable to embolize
Ferris,[30] 1981	25	Mixed	64% immediate; 56% (long-term)	
Uflacker et al,[12] 1985	5	TB in 76%	77% (immediate); 73% (long-term)	4% unable to embolize
Muthuswamy et al,[31] 1987	6	Active TB	83%	
Rabkin et al,[19] 1987	120	Mixed	91% (short-term); 78% (long-term)	8% failure, usually due to pulmonary artery source

*MH = massive hemoptysis.

whom bronchial embolization successfully controlled acute bleeding and Remy et al[28] report rebleeding in 15% of 41 patients controlled acutely with embolotherapy.

Prognosis of Massive Hemoptysis

Several series have examined features influencing mortality in patients with massive hemoptysis.[7,18] In a series reported by Corey and Hla,[7] with overall mortality of 34.5%, features associated with death included hypotension resulting from massive hemoptysis (72% vs 54% mortality in patients without hypotension), radiographic evidence of aspiration (44% vs 15% mortality), and underlying neoplasm as the cause of hemoptysis (72% vs 20% mortality). Importantly, the death rate was 6-fold higher in patients coughing > 1,000 mL blood/24 hours than in patients with smaller volume hemoptysis (58% vs 9% mortality). These observations are supported by Garzon and Gourin[18] who analyzed outcome in 75 patients expectorating > 1,000 mL/24 hours, 91% of whom underwent surgery. Overall mortality was 18% in this series, and features associated with mortality included large volumes of coughed blood (> 450 mL/12 h) and aspiration of blood to the contralateral lung. Though the number of patients in this series was small and the spectrum of causes of massive hemoptysis was narrow (eg, 71% of patients had tuberculosis), mortality was not influenced by patients' age, gender, etiology of bleeding, or administration of preoperative antituberculous therapy.

Clinical features that exclude patients as surgical candidates (eg, inadequate pulmonary function, debilitated states, bilateral pulmonary bleeding sources, metastatic cancer) are also highly associated with increased mortality rates in patients with massive hemoptysis. In a series of 67 patients bleeding > 600 mL/48 hours reported by Crocco et al,[6] 75% of inoperable patients died versus 40% of operable patients, with or without surgery. Mortality rates in inoperable patients were 46% and 71%, respectively, in series by Corey and Hla[7] and Garzon and Gourin[18] versus 16% and 18% in operable patients. That this mortality difference reflects the underlying clinical features rather than the impact of surgery is suggested by a lower mortality rate in operable patients *not* undergoing surgery (11% mortality) than in operable patients having lung resections (50% mortality).

Diagnosis of Hemoptysis

Proper management of patients with massive hemoptysis requires correct diagnosis and localization of the bleeding source. First, because hemoptysis can be mistaken for hematemesis, initial evaluation must distinguish between a gastrointestinal and a pulmonary bleeding source. As outlined by Lyons,[15] distinguishing features between hemoptysis and hematemesis are that blood from the lung is (1) coughed versus vomited, (2) partly frothy versus not frothy, (3) alkaline versus acid (unless hematemesis is so massive that stomach acid is neutralized by blood), and (4) mixed with pus, organisms, and macrophages versus sometimes admixed with food.

Also, patients with hemoptysis report gurgling in the chest and may localize a bleeding site in the thorax[2] versus experiencing nausea and vomiting, as is more typical in upper gastrointestinal bleeding.

Once the lung is established as the source of bleeding, attention turns to localizing the bleeding because treatment includes placing the bleeding lung in the dependent position and because successful use of topical therapy (eg, bronchial balloon tamponade), bronchial embolization, and/or surgical resection requires accurate knowledge of the bleeding source. Using all available methods, overall success rates for localizing bleeding have been 75-93%,[32] with 95% success in lateralizing the bleeding source without more specific lobar localization.[25] The yield of individual strategies for localizing the bleeding source has been assessed by Pursel and Lindskog[2] in a series of 105 patients with hemoptysis. Patient self-assessment was least useful, offered by only 10% of patients, of which 30% were inaccurate. Clinical examination correctly localized the bleeding source in 43% of patients but was inaccurate 2% of the time. Chest roentgenogram was correct in 60% of all 105 patients, and no instance of inaccuracy was found. Finally, bronchoscopy during active bleeding was most accurate (86% of evaluations) but was possible in only a minority (20%) of all patients.

Although most investigators advocate bronchoscopy to help localize bleeding, whether to perform bronchoscopy immediately (eg, within 48 hours of acute bleeding) or more remotely remains controversial. Table 7 presents the two available studies comparing early versus delayed bronchoscopy[2,33] that show higher rates of successful localization with early bronchoscopy. However, careful analysis of the diagnostic impact of bronchoscopy has shown that while active bleeding and the site of bleeding are visualized more commonly with early versus delayed bronchoscopy (34% vs 11%, respectively), the timing of bronchoscopy rarely alters the suspected cause of bleeding or overall patient management.[33] Nonetheless, to the extent that accurate localization is the keystone of managing massive hemoptysis and certainly can impact greatly on treatment, I favor early bronchoscopy. Whether to use a rigid or flexible bronchoscope has also been debated, but in the absence of a head-to-head comparison, no firm conclusion can be offered. Currently, the choice reflects the user's experience; the trade-off is between better suctioning with the rigid instrument and greater access to smaller airways with the flexible bronchoscope. The choice of instrument has been extensively summarized by Neff:[35]

> In cases of massive hemoptysis, the use of a rigid bronchoscopy for emergency examination seems more advantageous than using a bronchofiberscope. The selection of which scope to use for hemoptysis seems less important, however, than the close active communication between the experienced pulmonary physician and the skilled thoracic surgeon.

Besides plain chest radiography and bronchoscopy, three other imaging modes have been investigated for localizing the source of hemoptysis: computerized tomography of the chest; radionuclide studies using technetium sulfur colloid (^{99m}Tc-sulfur colloid) or red cells labeled with ^{59}Fe, ^{51}Cr, and ^{99m}Tc; and angiography of the bronchial or pulmonary arteries.

Only a single study has specifically examined the diagnostic impact of chest computerized tomography (CT) in patients with hemoptysis.[36] To assess whether chest CT provided unique diagnostic or management information, Haponik et al[36] performed plain chest radiographs, chest CT scans, and flexible fiberoptic bronchoscopy in 32 patients with hemoptysis of various causes. Computerized tomography scans provided unique imaging information in 47% of patients (15/32), including 10 patients in whom the plain film was normal. However, in only 2 patients did CT provide diagnostic information not available after bronchoscopy, leading to altered treatment in only 1 patient (3%). The authors conclude that, although CT markedly enhances radiographic diagnostic yield, the management impact of CT is more meager and "does not support the routine use of CT in evaluation of patients with hemoptysis."[36]

Radionuclide techniques using injection of ^{99m}Tc-sulfur colloid or isotope-labeled red blood cells have been evaluated in a small number of patients, as summarized by Haponik.[37] In general, availability and practical constraints may limit the usefulness of radionuclide studies in evaluating patients with massive hemoptysis, especially for techniques requiring preparation by labeling erythrocytes. In the scant experience with patients with massive hemoptysis, ^{99m}Tc-sulfur colloid has been injected intravenously with image collection over the subsequent 60 to 90 minutes. Dog studies suggest that this technique (using 4 and 15 mCi) can detect bleeding at rates as low as 0.1 mL/min.[38]

Table 7. A Summary of Available Studies of Delayed vs Immediate Bronchoscopy for Hemoptysis

First Author (Year)	n	Bronchoscopy	Yield of Localization of Bleeding	
			Delayed	Early
Pursel,[2] 1961	105	Rigid	52%	86%
Smiddy,[32] 1973	71 (active)	Flexible	NS*	93
Gong,[33] 1981	129	Flexible	11%	34%
Bobrowitz,[5] 1983	25	Flexible	NS	86%
Rath,[34] 1973	31	Flexible	NS	68%
Corey,[7] 1987	59	NS	NS	39%

*NS = not stated.

although detection is erratic and sometimes unsuccessful at bleeding rates 4-fold higher. Haponik has administered ^{99m}Tc-sulfur colloid (10 mCi) to 10 patients with hemoptysis deemed life-threatening and of uncertain pulmonary source.[37] Scans consistent with localized bleeding were obtained in 5 patients, 4 of whose bleeding was more precisely localized by closely timed flexible bronchoscopy. Bleeding was lateralized to the right side in the fifth patient whose bleeding site was not confirmed by other means. Overall, the limited impact of radionuclide techniques in the few patients reported suggests that routine use cannot be endorsed. Currently, use of ^{99m}Tc-sulfur colloid imaging in patients with massive hemoptysis should be confined to the usual clinical circumstance when the patient is relatively stable and bronchoscopy or chest CT is unavailable or nondiagnostic.

Treatment of Massive Hemoptysis

General principles for treating massive hemoptysis are outlined in Table 8. Localizing or, at least, lateralizing the bleeding is important to permit placing the bleeding lung down and to avoid contralateral aspiration, an event associated with increased mortality in massive hemoptysis.[18] Volume and coagulation management, supplemental oxygen, and general measures to prevent dislodging formed clots (eg, cough suppression and avoidance of maneuvers that raise intrathoracic pressure) are clinically sensible and have been widely adopted.

Table 8. General Principles for Medical Management of Massive Hemoptysis*

Localize bleeding
  History/physical exam
  Radiology
  Bronchoscopy (with topical therapy if possible)
Bleeding lung down
Volume/clotting management
  Fluid resuscitation with large-bore access
  Correction of any coagulopathy
  Administration of intravenous vasopressin
  Record of volume of hemoptysis
Oxygen therapy
General measures
  Cough suppression
  Stool softeners (to prevent straining)

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*Adapted from Reference 31.

In addition to these general maneuvers, more specific maneuvers are directed at the site of bleeding itself. These specific treatments can be categorized as shown:

- Topical treatments
  - Balloon bronchial tamponade
  - Topical coagulants (vasoconstrictors—eg, topical epinephrine)
  - Cold saline lavage
  - Radiation therapy
    - X-irradiation
    - Laser photocoagulation (Nd-YAG laser)
  - Positive end-expiratory pressure
- Pharmacologic maneuvers
  - Intravenous vasopressin
- Angiographic occlusion of source vessels
  - Bronchial artery embolization
  - Pulmonary artery embolization/occlusion
  - Embolization of other systemic arteries
- Surgical treatment
  - Lobectomy
  - Pneumonectomy
  - Cavernostomy

Topical Treatments

Although most investigation focuses on the efficacy of embolization and the need for surgery versus conservative management, topical treatments are important adjunct maneuvers in massive hemoptysis, especially because topical treatment can be administered during initial bronchoscopy for localizing bleeding.

In keeping with the time-honored treatment of upper gastrointestinal bleeding, cold saline lavage has been used acutely to stop bleeding in massive hemoptysis. In a single uncontrolled trial, Conlan and Hurwitz report that bronchial irrigation with 50 mL aliquots of cold (4°C) normal saline was successful in arresting bleeding in 12 patients with massive hemoptysis ($\geq$ 600 mL/24 h).[39] The average volume of saline required was 500 mL (range 300-750 mL) and the presumed mechanism is topical vasoconstriction with diminution of blood flow and thrombotic obstruction of distal vessels. Topical epinephrine (1:20,000) is used for similar reasons,[40] although the benefits for massive hemoptysis are uncertain and a structured trial has not been published. Finally, two reports in the Japanese literature[41,42] and a single report on 10 patients by Tsukamoto et al[43] describe the use of topical coagulants in patients with massive hemoptysis. Applying a topical solution of thrombin (5-10 mL of 1,000 units/mL), Tsukamoto et al report substantial efficacy in 50% of patients (no recurrence within 14 days), partial efficacy in 1 patient (hemoptysis recurred between 24 hours and 14 days after treatment), and failure to control bleeding in 40% of patients. Using a fibrinogen-thrombin solution (5-10 mL of a 2% fibrinogen solution) was very effective in 67% of patients (6/9), and partially effective in 33%. The authors contend that topical coagulant therapy "has efficacy equivalent to bronchial artery embolization," but lack of any controlled comparison and the paucity of available studies advise the reader to reserve judgment.

Mechanical topical measures, such as endobronchial tamponade, have been more widely adopted than topical drug treatments. Although bronchial tamponade has been used in conjunction with endotracheal intubation in exsanguinating hemoptysis, tamponade by placing bronchial balloons has been performed as a stand-alone measure in nonintubated patients.[44,45] Balloon tamponade involves occluding the bleeding airway by inflating a Fogarty catheter (4-Fr for segmental bronchi, up to 14-Fr for the left main-stem bronchus), which is left in place for 24 hours (after removing the bronchoscope over the catheter) and then removed.

Experience with bronchial balloon tamponade has been reported in two small series[44,45] in which bronchial balloon tamponade controlled bleeding acutely in all 10 patients with massive hemoptysis (≥ 300 mL/24 h), none of whom died from hemoptysis or experienced recurrent bleeding (without surgical resection) for up to 9 months of follow-up. In 4 patients with cystic fibrosis and massive hemoptysis, Swersky et al achieved acute control of bleeding, but bleeding recurred in 3 of their 4 patients.[45] No complications of endobronchial tamponade were noted in either series. Overall, though the reported experience is limited, the universal reported success with controlling acute bleeding and the lack of reported risk have caused endobronchial balloon tamponade to be widely used.

Other topical therapies for massive hemoptysis include external beam X-irradiation and laser photocoagulation with a neodymium yttrium aluminum garnet (Nd-YAG) laser, both of which have been described in a few case reports. External beam X-irradiation (2,000 rads in 5 fractions over 7 days) was associated with cessation of massive hemoptysis (450 mL/36 h) over 3 days after failed embolotherapy in a patient with an aspergilloma.[46] Recurrence of hemoptysis 2 months later again slowed after administering an additional 1,000 rads. Although X-irradiation seems sensible based on the vascular effects of radiation (ie, swelling, necrosis, endothelial cell hyperplasia causing thrombosis and fibrotic occlusion of vessels), judgment about the efficacy of X-irradiation must await further data.

Laser photocoagulation has been used to achieve hemostasis in patients with massive hemoptysis from airway neoplasms. For example, Edmonstone et al reported cessation of airway bleeding after applying 1,398 joules of Nd-YAG laser energy to an airway tumor.[47] However, failure of laser therapy to control bleeding due to an hemangiopericytoma has also been described.[48] While the sparse experience with Nd-YAG laser for massive hemoptysis precludes definitive recommendations, use of the Nd-YAG laser should be confined to centers at which it is available and to patients stable enough to permit laser therapy (ie, those not requiring immediate bronchial embolization and/or surgery).

Applying positive end-expiratory pressure has also been advocated as a 'topical' therapy for massive hemoptysis, especially in instances when the pulmonary artery is the source of bleeding and the patient is intubated (ie, in the operating room or intensive care unit). As noted by Noseworthy and Anderson[49] and Scuderi et al,[50] PEEP putatively lessens bleeding by lessening the pressure gradient between the pulmonary artery and airway. Specifically, PEEP is thought to be effective by compressing the pulmonary artery by increased intrathoracic pressure, decreasing pulmonary arterial blood flow and the transmural pressure gradient across the pulmonary artery wall, and decreasing the pressure gradient between the pulmonary artery and airway by raising airway pressure.

Available experience with PEEP for massive hemoptysis is scant and confined to two reports of 3 intubated patients in whom pulmonary arterial bleeding stopped when PEEP (20 cm H_2O) was applied. Though support for using PEEP to stop massive hemoptysis is limited, reasonable guidelines for considering PEEP in massive hemoptysis are as follows:

(1) Low-pressure pulmonary arterial bleeding is the presumed source of massive hemoptysis (eg, Swan-Ganz perforation of the pulmonary artery), and

(2) the patient is already intubated to control the massive hemoptysis (ie, in the operating room or intensive care unit).

In the majority of instances, when massive hemoptysis results from the bronchial arteries or other systemic arteries and the patient is not already intubated, PEEP is not an appealing treatment option.

Pharmacologic Maneuvers

Besides such topical treatments as cold saline lavage, bronchial balloon tamponade, and epinephrine, intravenous vasopressin has been used as a systemic pharmacologic treatment for massive hemoptysis. As early as 1911, Wiggars[51] observed that pituitary extract could arrest pulmonary bleeding, presumably by lowering pulmonary vascular pressure. Subsequent use of pituitary extract[52] and of vasopressin have shown efficacy in massive hemoptysis from bronchiectasis, tuberculosis, and lung abscess, all conditions in which pulmonary bleeding may arise from the bronchial arteries. As with its use in gastrointestinal bleeding, the hemostatic effect of vasopressin in hemoptysis may reflect its activity as a potent systemic arterial vasoconstrictor. The recommended dose of intravenous vasopressin for hemoptysis is 0.2-0.4 units/minute as a continuous infusion, and vasopressin infusion is best regarded as a temporizing measure to control bleeding until more definitive measures are implemented. Importantly, Remy et al[28] have pointed out that bronchial arterial constriction by vasopressin can preclude attempts to embolize the bronchial arteries by preventing angiographic visualization of the vessel.

Embolotherapy of Bronchial Arteries, Other Systemic Arteries, and Pulmonary Arteries

Besides topical therapies of massive hemoptysis, angiographic occlusion of source vessels (embolotherapy) and surgical resection are options.

Several lines of evidence suggest that the bronchial arteries are the usual source of massive hemoptysis:

1. Injection studies show that bronchial arteries feed chronically inflamed regions of lung;
2. bronchial arteries feeding inflamed lung enlarge in diameter (from 2 mm up to 6 mm);
3. in contrast, pulmonary arterial branches supplying inflamed lung often thrombose early; and
4. anastomoses between bronchial arteries and pulmonary arteries enlarge in chronic inflammatory conditions, exposing thin-walled low-pressure vessels to systemic pressures.

Perhaps the most compelling evidence that bronchial arteries are the usual source of massive hemoptysis is that embolization of the bronchial arteries has arrested bleeding acutely in patients with massive hemoptysis from many causes (64% to 100% success, Table 6[27-31]). In the largest available single series, Rabkin et al[19] reported acute cessation of bleeding in 91% of 306 patients undergoing bronchial embolization. In another large series of bronchial embolization, Remy et al[28] reported cessation of active, massive bleeding in 41 of 49 (84%) patients treated by injecting 1- to 2-mm pellets of a microencapsulated absorbable gelatin sponge into the bronchial arteries. In patients with both massive (n = 35) and recurrent (n = 29) hemoptysis, Uflacker et al reported 77% immediate control of hemoptysis after injecting Gelfoam particles (2 × 2 mm and 10 × 3 mm) into the bronchial arteries.[13]

The high rate of longer term success, measured by freedom from recurrent bleeding, suggests that bronchial arteries are the usual source of massive hemoptysis. Table 6 presents long-term success rates from 54% to 78% in several available series.[27-31] More specifically, Remy et al[28] noted that only 14.6% (6/41) of the patients whose active bleeding was successfully arrested after bronchial embolization experienced recurrent bleeding over the follow-up interval (2-7 months). Uflacker et al[12] reported recurrent hemoptysis in 30.3% of patients treated by bronchial embolization alone.

Putative causes of remote rebleeding after initial control include progression of the underlying disease, development/enlargement of systemic collateral arteries, and recanalization of the embolized vessels. Partial or complete recanalization of bronchial arteries has been observed in 3 of 6 patients re-studied by Uflacker et al[13] after hemoptysis recurred.

Reasons that bronchial embolization may fail to control bleeding acutely include (1) the embolization is technically unsatisfactory (ie, vessels cannot be visualized or if seen cannot be embolized) and (2) the sources of bleeding are vessels other than the bronchial arteries (ie, collaterals to the lung branching from systemic vessels other than the bronchial arteries such as the subclavian, axillary, phrenic, or coronary arteries or from the pulmonary artery).

Features identifying bronchial arteries as appropriate targets for embolization include vessel hypertrophy (ie, increased size, number, and/or tortuosity), systemic-to-pulmonary shunting, aneurysm formation, and/or dye extravasation, which is uncommon (seen in 3 of 75 patients in one series[13] and 22% of patients in another[19]). Despite these target features, technical failure occurs in 4-13% of attempted bronchial embolizations[13,19,28] because of inability to visualize or cannulate the vessel or because takeoff of the anterior spinal artery (Adamkiewicz) makes attempting embolization imprudent. As noted above, concomitant administration of intravenous vasopressin can cause spasm of the bronchial artery and preclude visualizing and/or cannulating the vessel.[28]

Pulmonary bleeding will persist after bronchial embolization if the source of the bleeding is not the bronchial artery. In a series of 20 consecutive patients with hemoptysis from many causes treated by bronchial embolization, Keller et al[14] have reported that control of bleeding required embolization of other systemic collateral vessels in 9 patients (45%). Sixteen subclavian or axillary arteries and three phrenic arteries required occlusion to achieve hemostasis in this series.

Though less frequently the source of hemoptysis than the bronchial arteries, the pulmonary arteries are another important bleeding source and should be examined angiographically if bleeding persists after bronchial and systemic collateral arteries have been treated. Remy et al[28] reported that among 72 patients referred for embolization and studied with both bronchial and pulmonary angiograms, the pulmonary artery was the source of bleeding in 6 (8.3%). Erosive pseudoaneurysms of the pulmonary artery were observed in 5 of these patients, associated with aspergilloma (n = 1), cavitary tuberculosis (n = 12), and pyogenic abscess (n = 12). Similarly, Rabkin et al[19] have reported that bronchial embolization failed to arrest pulmonary bleeding in 28 of 306 patients referred for angiography. In 26 of 28 (8% of the total 306), bleeding was only stopped by subsequently embolizing the pulmonary artery. Finally, Bredin et al[53] have reported a patient with tuberculous bronchiectasis whose bleeding slowed after bronchial emboli-zation but did not stop completely until the pulmonary artery was occluded by inflating the Swan-Ganz catheter balloon.

When hemoptysis accompanies placement of a Swan-Ganz catheter, pulmonary artery tear, rupture, or infarction are obvious considerations. Putative risk factors for pulmonary artery rupture by a flow-directed catheter include (1) advancing the catheter too far distally (as may happen when a catheter that is placed preoperatively migrates distally as the lung is deflated for chest surgery); (2) inflating the balloon when the catheter is too distal or overinflating the balloon;[54] and (3) wedging the catheter with the balloon incompletely inflated, exposing the catheter tip beyond the balloon like a spear.[55]

Acute management of pulmonary artery tears includes balloon inflation for vessel tamponade,[53] embolization of the pulmonary artery,[28] or emergency surgery with suture occlusion of the proximal pulmonary artery or lung resection.[54,55]

Although somewhat controversial, tuberculosis is another setting in which the pulmonary artery must be suspected as the source of hemoptysis. Rasmussen identified aneurysms of the pulmonary artery in the walls of tuberculous cavities in 11 postmortem examinations in 1868,[56] an observation that has lent his name to tuberculous pulmonary arterial aneurysms.[13,56] Subsequent observers have confirmed these findings,[57,58] which have been disputed by Cudkowicz,[59] who suggested that the bronchial arteries were the true source of the aneurysms traced by Rasmussen to the pulmonary arteries and who observed thrombosis of the pulmonary artery radicals in five postmortem examinations.

Despite the possibility of a pulmonary arterial source of bleeding in tuberculosis, the high rate at which bronchial embolization successfully controls acute bleeding[31] (Table 6) suggests that bronchial angiography should be undertaken before pulmonary angiography.

Although highly successful in controlling acute bleeding, bronchial angiography may be associated with complications. Table 9 reviews the reported complications that may result from the catheterization approach itself, the expected consequences of occluding the bronchial artery, unexpected consequences (eg, bronchial necrosis), or complications due to occluding other, unintended arteries. Of greatest concern, but reportedly rare,[13,19] is the risk of occluding the anterior spinal artery, which may emanate from the right intercostal bronchial trunk in up to 5% of patients.[11] Inadvertent embolization of the anterior spinal artery can cause transverse myelitis or paralysis if the occlusion is complete. Finally, occlusion of other aortic tributaries can cause tissue ischemia or infarction. For example, in the series by Remy et al.[28] 1 of 104 patients undergoing bronchial embolotherapy experienced necrosis of 15 cm of small bowel. Occlusion of the anterior tibial artery with resultant claudication, transient right arm numbness presumably related to inadvertent reflux of embolizing material into the subclavian artery, and renal embolization have been reported. Fortunately, the frequency of 'ectopic' embolization in reported series is very low (< 2% of combined series).[13,19,28]

Table 9. Reported Complications of Bronchial Embolization

- Complications of catheterization
  - Aortic intimal tear
- Expected complications of bronchial artery ischemia/infarction
  - Cough
  - Chest-wall pain (universal)
  - Fever (universal)
- Unexpected sequela of bronchial artery ischemia/infarction
  - Bronchial necrosis (ethanol injection)
- Complications due to 'ectopic' embolization
  - Spinal cord ischemia/infarction (artery of Adamkiewicz)
    - Transverse myelitis (noted but not reported to occur)
    - Paralysis
  - Ischemia/infarction of aortic tributaries (< 2% combined series)
    - Leg vessels
    - Renal
    - Bowel

Overall, bronchial embolization has become a mainstay in treating life-threatening hemoptysis because acute bleeding can usually be arrested, because rebleeding after an initial treatment is relatively unlikely, and because the procedure is generally safe. Bronchial embolotherapy is an attractive first-line therapy that can preempt the need for surgical resection.

Surgical versus Medical Therapy for Massive Hemoptysis

Surgical approaches for massive hemoptysis include lung resection (ie, lobectomy or pneumonectomy) and nonresectional treatment (ie, cavernostomy to pack the bleeding site); the latter is reserved for patients deemed incapable of withstanding lung resection and is infrequently performed.

To the extent that surgery removes the bleeding lesion and the source vessels, resection is successful in arresting bleeding acutely and preventing recurrence, as long as the disease is localized to the resected segment and does not progress. An obvious exception would be massive hemoptysis from diffuse bilateral bronchiectasis, when resection of one lobe might not prevent future bleeding from other areas of involved lung.

Table 10 reviews available series that report mortality with medical and surgical management of patients with massive hemoptysis. All available studies are observational series because a randomized controlled trial of medical versus surgical therapy of massive hemoptysis has not yet been conducted. Comparison of series that span two decades reveals a striking range of mortality rates (ie, for surgical resection 0.9%[60] to 50%[7] and for medical management 1.6%[5] to 85%[60] in various series). Among these six series, conclusions regarding the efficacy of surgical versus medical therapy also vary widely. Four series report higher medical than surgical mortality, and the two most recent series[5,7] report lower mortality with medical management than with resection. In an attempt to understand these disparate conclusions, analysis of the available series is appropriate and shows major differences among the designs of available studies. Surgical investigators have authored the two series

Table 10. Outcome of Surgical (S) vs Medical (M) Treatment of Massive Hemoptysis

First Author (Year)	n	Mortality (%) Surgical	Mortality (%) Medical	Comments
Yeoh,[24] 1967	13 S, 43 M	15	23	Not randomized, no arterial embolization
Crocco,[6] 1968	32 S, 35 M	19	54 (78*)	Not randomized, no arterial embolization
Gourin,[20] 1974	57 S, 5 M	18	80†	Not randomized, no arterial embolization
Sehhat,[60] 1978	106 S, 40 M	0.9	80 (85*)	Not randomized, no arterial embolization
Garzon,[9] 1982	24 S	25	—	Exsanguinating hemoptysis (≥ 1,000 mL blood at ≥ 150 mL/h)
Bobrowitz,[5] 1983	31 S, 82 M	13	22 (1.6*)	Not randomized, no arterial embolization
Corey,[7] 1987	4 S, 55 M	50	29 (11*)	Not randomized, no arterial embolization

*Surgical candidates managed medically.

†Medically managed, deemed inoperable.

with the largest survival difference favoring surgery and the highest medical mortality rates (80-85%);[20,60] unlike the other series, the two surgical series also report the highest proportion of patients offered surgery (91.9%[20] and 72.6%[60] of patients, respectively) and the highest proportion of patients deemed surgical candidates. Though difficult to specify, it is possible that strategies for allocating patients to surgery rather than medical therapy may have differed in these series from other series collected by nonsurgeons, raising the possibility that selection bias may account for some of the disparate conclusions.

Because being considered inoperable suggests a poor prognosis of itself, appropriate interpretation of medical mortality rates among series requires comparison of mortality rates among patients deemed operable but managed medically. Mortality rates of such operable but medically treated patients are presented in parentheses in Table 10, and striking variation is present between recent series[5,7] and older, surgical series.[20,24,60] That mortality rates are lower (1.6-11%) among operable patients treated medically than among all medical patients (which includes some inoperable patients) is clinically sensible, while the higher mortality rates among operable medical patients than all medical patients in the series by Crocco et al[6] and Sehhat et al[60] is difficult to explain. Overall, except for a few outliers,[7,60] available series suggest a generally stable, low surgical mortality rate with a generally declining medical mortality rate among series, which is impressively lower among operable but medically managed patients (1.6-11% mortality).[5,7] Furthermore, to the extent that none of the series in Table 10 includes effective medical treatments like topical therapy (eg, bronchial balloon tamponade) or embolization as part of the medical regimen offered, it is likely that the benefits of state-of-the-art medical management are underestimated by these series. A recent preliminary report by Shamji et al is especially informative.[25] Of 22 patients with massive or life-threatening hemoptysis seen at the University of Ottawa over 6 years, 2 died acutely and 20 survived the acute episode. Bleeding stopped spontaneously in 6 of 20 surviving patients, the remainder of whom underwent selective bronchial occlusion (13 patients) or bronchial embolization (6 patients). Twelve of the progressively bleeding patients underwent surgery with a 92% acute survival; and 8 patients were managed medically, 7 of whom survived (88%). Overall, acute survival was 82% in this series, reflecting the benefits of state-of-the-art medical therapy and modern surgery for patients who bled progressively despite medical therapy.

In this author's view, the high rate of acute success and low reported risk with bronchial embolization recommends embolotherapy as first-line treatment for patients with recurrent massive hemoptysis or unremitting hemoptysis on initial presentation. Surgery is best reserved for operable patients when

- Bronchial embolization is not available or is technically impossible (eg, anterior spinal artery takeoff from the bronchial artery, bronchial artery not visible angiographically).

- Bleeding continues despite attempted embolization.
- The volume of hemoptysis requires definitive initial treatment (ie, > 1,000 mL/24 h or is associated with hemodynamic or major respiratory compromise).
- The cause of massive hemoptysis is unlikely to be arrested by bronchial embolization (pulmonary artery perforation by Swan-Ganz catheter, mycetoma with profuse systemic collateral arterial supply).

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Stoller Discussion

Porter: In the experience that you quoted with vasopressin and desmopressin (I know those are probably very small numbers) but were results any better with desmopressin—because it has the possible additional effect of being a coagulant or of activating platelets?

Stoller: Yes, the use of desmopressin, or DDAVP, has gained popularity, as you know, in renal failure and in preventing bleeding after kidney biopsy, taking advantage of its ability to mobilize Von WilleBrand's factor. It is a less potent vasoconstrictor. It is an arginine-substitute vasopressin, so that it is less effective as a systemic vasoconstrictor and more effective as a 'coagulant,' as you put it. The numbers are small and totally anecdotal, but I think if one had to choose, one would certainly use intravenous vasopressin over desmopressin.

Wilson: How does time enter into the algorithm that you presented? Many of these series as you pointed out are very small and have occurred over the course of many decades. Now that we have aggressive angiography services available essentially 24 hours a day, is this medical/surgical approach now compressed into a 24-hour period or is it a week period of time? Has that influenced the figures, as opposed to what may have happened 10 years ago?

Stoller: That's an excellent question, and it's a variable not addressed in much of the literature. Very few of the available reports present the time interval between the onset of disease and the intervention, as it regards success or failure. But in my own view, one needs to make decisions fairly promptly with regard to presentation, and the initial assessment would include scrolling through the differential diagnosis to decide whether this is likely to be from a pulmonary or bronchial artery, a mycetoma, TB, Bechet's, a pulmonary or Swan-Ganz catheter. I mean those are the kinds of questions in the algorithm. If the patient is at risk for soiling the contralateral lung, I think it may be appropriate to intubate the patient and perhaps to call the bronchoscopist or take advantage of balloon tamponade as a temporizing measure en route to the angiography suite. If the angiographer is not available, if angiography is not possible, or if it is not an option or is somehow technically problematic or not going well, one would have a low threshold for surgical resection within the next 24 hours—because patients that don't do well die soon. That's the message from Yeoh's paper[1] and from Bobrowitz's paper.[2] They don't linger around, coughing up 150 mL every day in a systematic way. They cough then and there, and the time frame is compressed into hours, I think, not over days or weeks. With regard to what happens after the acute control, that's also problematic, but I think fewer and fewer patients will escape without some high tech intervention, whether it be bronchial embolization or surgical resection. A possible exception is patients with CF, for whom the approach has been somewhat more conservative, with acceptable results, as shown by Stern's series.[3] But I think the time frame is short for the decision making.

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Reines: You didn't mention the use of the Carlen's tube. In our institution, with massive hemoptysis, and with very good pulmonary anesthesiologists, that's an early option. The other subset you didn't mention is a subset that Dr Hurst and I deal with, and that's trauma patients. They were not on any of your series as a cause of either hemoptysis or massive hemoptysis. In my practice, that plus tracheoinnominate fistulae are surgical causes of hemoptysis.

Stoller: Those are both excellent points. With regard to Carlen's tubes, really only a single series, Gourin and Garzon[1] have addressed the actual instrument that's best to use in the isolation of an airway. Actually, the literature has moved away from Carlen's tubes because of the difficulty of keeping both airways cannulated and because of the small caliber of the Carlen's tube at the takeoff, which prohibits good suctioning and certainly prohibits bronchoscopic passage through the Carlen's tube. What literature that does exist reflects the strategy of intubating the main airway and cannulating distally, as I showed, using bronchial balloon tamponade to cannulate the bleeding lung distal to a standard endotracheal tube in the proximal trachea. Inflating the balloon isolates the lung. There has been some experience with Robertshaw tubes that has been somewhat disappointing as well. It's important to avoid trying to intubate the right lung because that's been fraught with some difficulty. The Carlen's vs bronchial embolization experience from Gourin and Garzon[1] suggests that the latter strategy is better than using a Carlen's tube. With regard to acute traumatic management, some of the patients in Rabkin's series of 306 patients had acute trauma, in which the angiographer was called to the emergency room to manage the acute bleeding patient with bronchial angiography,[2] but there's very little literature about the acute traumatic management of massive hemoptysis. So, I think it's wide open for someone to make a thoughtful contribution to the literature.

1. Gourin A, Garzon AA. Operative treatment of massive hemoptysis. Ann Thorac Surg 1974;18:52-60.
2. Rabkin JE, Astafjev VI, Gothman LN, Grigorjev YG. Transcatheter embolization in the management of pulmonary hemorrhage. Radiology 1987;163:361-365.

Haponik: I think you have done a fabulous review, given the heterogeneity of the literature—both regarding time and the spectrum of diseases and the differences in management approaches to these patients. At an ACCP meeting a few years ago, we had the opportunity to survey practicing chest clinicians about their perspectives on these issues.[1] It's remarkable the degree to which the algorithm you outlined is really consistent with current practice patterns by pulmonologists. Despite the recognized advantages of a rigid-bronchoscope approach, most practitioners now are leaning (resulting from their training perhaps) toward the use of a fiberoptic instrument in the ways to which Jamie (Stoller) has alluded. Practice by most individuals also reflects his recommendation for placement of a large-bore endotracheal tube, rather than use of a Carlen's tube. Even in prepared operating room settings, failure rates as high as 25% with insertion of Carlen's tubes and Robertshaw tubes have been recognized. And, as so often occurs, this catastrophic event takes place in the middle of the night when the initial management of this 'drowning' confronts perhaps the least experienced members of a management team. I'd like to ask you a question regarding the specific roles of respiratory care practitioners in this area. It's remarkable how little there is in the respiratory care literature regarding what to do in this clinical setting. How about the issues of positioning, administration of chest physiotherapy, inhalation therapy, and potential hazards in that regard, or what information really is available?

1. Haponik EF, Chin R. Hemoptysis: clinicians' perspectives. Chest 1990;97:469-475.

Stoller: Those are excellent points, and I neglected to cite the consensus or the questionnaire that you cited, which is an important statement from the chest literature. With regard to the respiratory therapist's contribution, I think it's important that the respiratory therapist be familiar with acute management issues of localizing bleeding and putting the bleeding lung down. It's not infrequent that the therapist is the only person actually thinking about the dynamics of where the blood's coming from and what to do. As simple a maneuver as putting the bleeding lung down does not occur to many people who are not used to thinking about the airway and the lungs. So in keeping with the theme of having the respiratory therapist as an on-site source of information, which is what Paul Mathews, Bob (Kacmarek), and Roger (Wilson) alluded to earlier, I think that therapists are the people who are in fact more expert in the acute management issues than many of the other people who are called to attend to the patient. In preparing this talk, I found relatively little from the respiratory care literature and our own literature regarding this.

Mathews: Concerning the role of the respiratory therapist in hemoptysis—which is spoken to in the literature related to contraindications of the therapies that we give, positive-pressure breathing therapies, and, I suppose, by extension, perhaps mechanical ventilation, chest physical therapy, and vibration. Do you have any thoughts for us on those issues? How contraindicated are these contraindications?

Stoller: Paul, that's an excellent question. There's very little with any scientific basis, but the standard advice is to avoid dislodging clots, to give cough suppressants and stool softeners in order to prevent the patient from straining at stool and increasing intrathoracic pressure, and to avoid chest physiotherapy. Some people are more aggressive about cough suppressants than others. There's very little in the way of rigorous guidelines, and yet my own sense of it is that avoiding aggressive chest physical therapy is a good thing to do. I think as we get more and more into therapist-driven protocols and more and more primary decision making by respiratory therapists (which is an important trend that I think is emerging), this will become an important role of the therapist in the acute setting.

Rodriguez: One of the areas in which the therapist encounters hemoptysis most often is in nasotracheal suctioning. Are there any comparisons that can be made from the management of large volume hemoptysis and this trauma-induced hemoptysis?

Stoller: I'll defer that question to Roger (Wilson), who taught me everything I know about upper airway management. I can't cite any literature in that regard.

Wilson: That's sort of a difficult analogy because it's like cutting your finger and cutting your arm off. I mean, what Jamie (Stoller) has tried to describe is life-threatening and dramatic. These are patients in whom urgency is the first order because they're truly exsanguinating, and they're being asphyxiated. They're in a much different category from the individual who has therapy-induced traumatic bleeding of the airway, both in terms of avoidance and in terms of the priorities of their management. It's really apples and oranges, and I don't think it's really fair to compare them.

Fanta: Jamie, does systemic hypertension contribute to or perpetuate bleeding from the bronchial circulation, and would you cautiously reduce blood pressure in someone with systemic hypertension and hemoptysis, including massive hemoptysis?

Stoller: That's a good question. I presume that there's some autoregulation of the bronchial arterial circuit, but I'm not aware of any literature. Obviously, if one lowers the blood pressure enough, one will compromise perfusion everywhere. The question is whether it would selectively affect the bronchial arterial circulation. I guess I can't answer that, because I'm not aware of that having been addressed in the literature. I'd be happy to hear comments from others.

Haponik: I've heard anecdotes regarding attempts to use vasodilators in the situation; but they are only anecdotes, and that's certainly not part of standard care. One other comment I'd allude to would be the importance of respiratory care practitioners' reminding the managing team to avoid anxiolytics. As was pointed out earlier this morning, until one has control of the airway, you're courting disaster in this patient population. Until we have an airway, effective cough is really the only defense this patient has, and too often the importance of that is disregarded by the managing clinician. The other patient type that I'd like to add to your list of potential patients for early surgery would be those with clear-cut, focal, resectable necrotic lesions. I think there are some anecdotal reports in the literature that support earlier surgery in these individuals, particularly patients with lung abscess, but, again, the data supporting that are treacherous to interpret.

Stoller: I think that's an excellent point, and perhaps one other subset would be patients presenting with hemoptysis in whom medical therapy is likely to be disappointing—for example, in a patient with bronchiectasis and *Mycobacterium avium-intracellulare* complex [MAI] in which the expected efficacy of 5, 6, 7 drug regimens is poor. Such a patient with localized disease and hemoptysis might be better managed, both from the point of view of his underlying disease and the acute presentation, with referral to a surgeon, rather than the attempt to temporize with another strategy.

Neonatal and Pediatric Airway Emergencies

John E Thompson RRT, Edward Farrell BS RRT, and Michael McManus MD

Introduction

This review is intended to inform and assist the respiratory care practitioner in being prepared to respond effectively during a pediatric or neonatal airway emergency. Equipment and techniques specific to the care of infants and children are described and common disease entities, such as meconium aspiration, congenital diaphragmatic hernia, tracheolaryngobronchitis (croup), epiglottitis, and foreign-body aspiration, are discussed. Familiarity with the relevant differences between adult and pediatric patients and the general therapeutic approaches to these differences should permit the therapist to provide timely and often lifesaving care.

Anatomy

Anatomic differences between the adult and the child are most apparent in the newborn infant and gradually disappear as the child reaches the school-age years. Although differences in size predominate, differences in anatomic orientation, structure, and physiology also contribute to the challenge of a difficult pediatric airway.[1]

Infants and young children are particularly vulnerable to airway compromise. With laminar flow, the flow of gas (volume/time) moving through a tube is directly proportional to the fourth power of the radius of the tube. Thus, a reduction in diameter by half reduces flow to 1/16 of its former level. To maintain the same flow requires a 16-fold increase in pressure—thus the work of breathing is greatly increased. When flow becomes turbulent, resistance and hence work of breathing increase even more.[2,3] Physiologic factors such as increased oxygen consumption, increased metabolic cost of breathing, decreased functional residual capacity (FRC), and increased closing volumes limit the oxygen reserves of pediatric patients (particularly infants) and compound their vulnerability to airway emergencies.[4]

Other than size, anatomic peculiarities of the pediatric airway include a large tongue (relative to the size of the oral cavity), a rostral and more anteriorly situated larynx (at the level of the third and fourth cervical vertebra, C3 and C4), angled vocal cord attachments, a curved and angled epiglottis, and a funnel-shaped larynx (with the limiting diameter being at the cricoid cartilage rather than the vocal cords). Rostral placement of the larynx (C3-4) allows the epiglottis to contact the soft palate thus permitting the newborn to suck and breathe simultaneously. Unfortunately, this also obligates the infant to nasal breathing and makes her more vulnerable to nasal obstruction from secretions, foreign objects, adenoidal tissues, and edema.[3] The soft, fragile, less adherent mucosal tissues of the infant nasopharynx may easily swell in response to infection or trauma (suctioning), thus producing serious airway obstruction. In the older patient, tonsillar or adenoidal hypertrophy or infection can

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Mr Thompson is Director, Respiratory Care Department, Children's Hospital, and Associate in Anesthesia, Harvard Medical School; Mr Farrell is Supervisor, Respiratory Care Department, Children's Hospital; Dr McManus is Associate Director, Multidisciplinary Intensive Care Unit, Children's Hospital, and Instructor in Anesthesia, Pediatrics, Harvard Medical School—Boston, Massachusetts.

A version of this paper was presented by Mr Thompson on October 3, 1991, during the RESPIRATORY CARE Journal Conference on Emergency Respiratory Care held in Cancun, Mexico.

Reprints: John Thompson, Respiratory Care Department, Children's Hospital, 300 Longwood Ave, Boston MA 02115.

lead to either complete or partial airway obstruction with obstructive sleep apnea being a common presentation.

The rostral and anterior larynx with its acutely angled vocal cords and funnel shape presents specific difficulties during intubation. Whereas neck extension in the adult tends to open the airway and facilitate intubation, extension of the more flexible neck of the infant actually worsens conditions by closing the intubation angle and, occasionally, kinking the trachea.[5] Optimal positioning (the so-called sniffing position) can usually be attained by simple supine positioning, with the infant's large head and prominent occiput providing the necessary lift. Endotracheal tube (ETT) passage through the vocal cords frequently may be hampered by the acute angle and anterior attachment of the cords. As the tip of the ETT contacts and 'hangs up' on these anterior attachments, manipulation of the larynx (with cricoid pressure) and the tube tip (by rotation and stylet placement) become necessary.

Finally, intubation requires tube selection appropriate to the diameter of the infant's cricoid cartilage rather than of the vocal cords. Because this area is not visible during simple laryngoscopy, the appropriateness must be judged indirectly by ease of passage and resultant air leak. A seal of sorts is produced between the ETT and the mucosa lining the cricoid, thus making a cuff unnecessary. However, this contact is itself traumatic and over time can produce ciliary denudation, inflammation, weakening of the cartilage, and eventual scarring. Thus, edema, mucosal eventration, subglottic stenosis, and granuloma formation become possible complications.[1]

In addition to normal anatomic differences, a myriad of pathologic differences can present themselves. Laryngomalacia, a common cause of infantile stridor,[6] involves the inspiratory collapse of abnormally floppy laryngeal tissues. Micrognathia, as in the Pierre-Robin malformation sequence, results in airway obstruction due to posterior displacement of the tongue. Macroglossia, as in the Beckwith-Wideman Syndrome, produces similar effects. Specific malformations such as encephaloceles, hemangiomas, oral lymphangiomas, and intrinsic tissue changes (as with the gangliosidoses) are common complicating factors in pediatric referral populations.

Neonatal Airway Management

The Apgar scoring system[7] has been a widely adopted standard for initial clinical assessment. Status is quantified through rapid analysis of the infant's color, heart rate, tone, respiratory drive, and response to stimulation at 1 and 5 minutes of life (Table 1). Although the 5-minute score correlates best with ultimate outcome, low scores at 1 minute (Table 2) alert the practitioner to the need for immediate intervention.[8] Ongoing ventilation

Table 1. Apgar Scoring Chart for Newborn Assessment*

Sign	Score 0	Score 1	Score 2
Heart rate	Absent	Slow (< 100)	≥ 100
Respiratory effort	Absent	Slow, irregular, hypoventilation	Good Crying lustily
Muscle tone	Flaccid	Some flexion of extremities	Active motion, well flexed
Reflex irritability	No response	Cry Some motion	Vigorous cry
Color	Blue, pale	Body pink Hands and feet blue	Completely pink

*Adapted, with permission, from Reference 7.

Table 2. Apgar Score Implications for Treatment*

Apgar Score	Presentation	Respiratory Intervention
8-10	No significant asphyxia	No treatment
5-7	Mild asphyxia	Tactile stimulation; 100% oxygen by face mask or free flow
3-5	Moderate asphyxia	Rapid stimulation and 100% oxygen bag-mask ventilation; restore FRC and spontaneous respirations
0-2	Severe asphyxia	100% bag-mask ventilation heart rate < 100 beats/min; intubation should be performed

*Adapted, with permission, from Reference 8.

requirements may be assessed by heart rate. A heart rate less than 100 beats per minute necessitates continued ventilation. The majority of infants can be resuscitated with stimulation, supplemental oxygen, and, on occasion, brief bag and mask ventilation. However, without timely intervention, an initially reversible situation can deteriorate to a more serious and often irreversible one. During initial stabilization of the newborn, the "five Hs" must be resolved: hypoxia, hypercarbia, hypotension, hypoglycemia, and hypothermia.

Resuscitation Equipment in the Delivery Room

Delivery room equipment for infant resuscitation should include a system for positive pressure ventilation (either self-inflating or gas-supply dependent), masks, ETTs, laryngoscopes, and suctioning paraphenalia.

Non-Self-Inflating Systems. The non-self-inflating systems (the so-called anesthesia bags) have their roots in the T-tube systems developed in 1937 by Ayre[9] for delivery of anesthesia to spontaneously breathing infants. In 1950, the system was modified by Rees[10] with placement of a double-ended bag to allow control of ventilation. A number of variations were developed later, and Mapleson[11] provided a classification system in 1954.

The essential elements of anesthesia or Mapleson systems are a T-piece, a corrugated reservoir tube, a non-self-inflating reservoir bag, a fresh gas source (essential) and, in Types A-D, a 'pop-off' valve (Fig. 1). In the simple Ayre's system (Type E) rebreathing is prevented by providing both a high fresh-gas flow (2-3 times minute ventilation) and a small reservoir (from which to draw when inspiratory flow exceeds fresh-gas flow). Instead of a pressure-relief, or pop-off, valve, the Mapleson F circuit employs a small hole at the end of the reservoir bag, which can be pinched closed. In our institution, this system is preferred because it allows the operator to control system leak level (pop-off) and thus system pressure. In all of these systems, the products of expiration are continuously flushed from the system (Fig. 2). Required fresh-gas flow may be initially approximated by the Nightingale formula (Table 3) for spontaneous respiration.[12] Mechanical ventilation still requires 2-3 times minute ventilation.

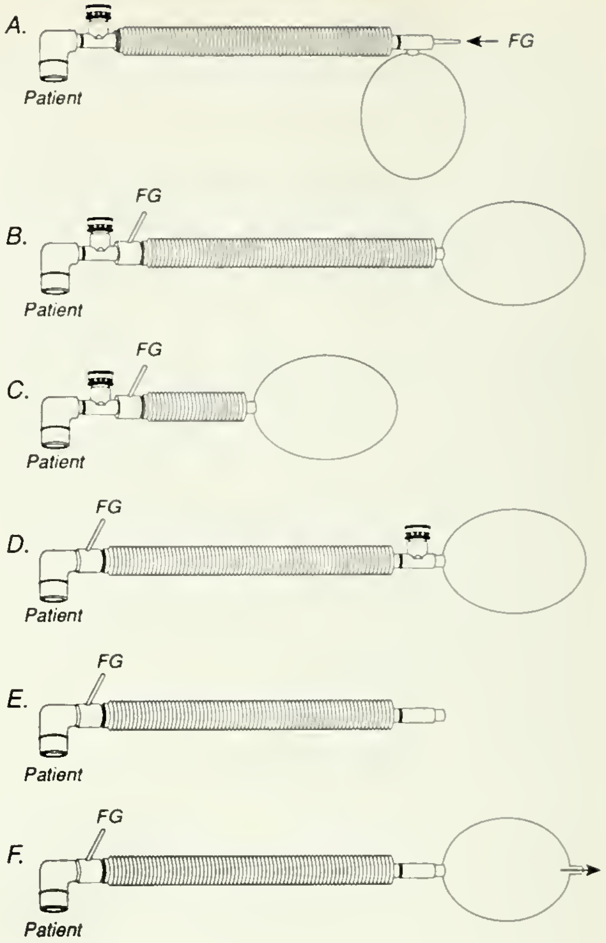

Fig. 1. Modified Mapleson classification system. FG = fresh gas. (Adapted from Reference 10.)

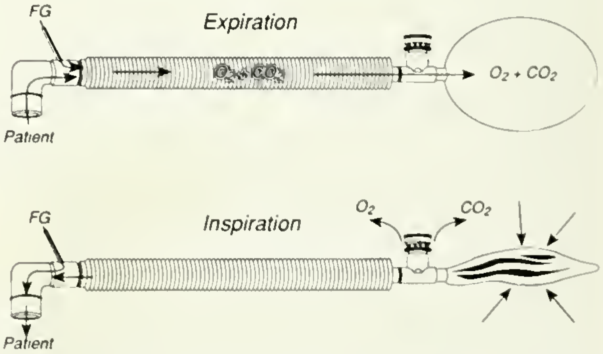

Fig. 2. Mapleson D system during expiration. Fresh gas (FG) flushes CO_2 and O_2 to reservoir bag. During inspiration, fresh gas is delivered to patient and contents of reservoir bag exits through pressure-relief ('pop-off') valve.

Table 3. Nightingale Estimates of Fresh Gas Requirements*

Patient Weight	Requirements
< 30 lb	Fresh gas = 3 L/min
30-80 lb	Fresh gas = 100 mL · min · lb^{-1}
> 80 lb	Fresh gas = 8 L/min

*Adapted from Reference 12.

When a reservoir bag is added to the circuit, pressure on the bag allows control of ventilation. Placement of the fresh-gas flow near the patient and of the pop-off valve near the bag (Mapleson D) allows control of ventilation with minimal rebreathing. However, rebreathing increases as fresh-gas flow declines and ventilation becomes inadequate. Other disadvantages of these systems include the requirement for an experienced user and the possibility of delivering excessively high inflation pressures (thus in-line manometry is recommended). Advantages include the abilities to deliver accurate (1) F_{IO_2}, (2) inflation pressure, and (3) PEEP, and to allow the delivery of (4) accurate inspiratory times. Rebreathing may be assessed and avoided through use of an end-tidal CO_2 monitor.

Self-Inflating Systems. Self-inflating systems are by far the most popular and simplest to use for emergency resuscitation.[13] Most neonatal bags contain volumes of 200-300 mL; however, some practitioners suggest slightly larger systems (400-500 mL) to allow 1-second, sustained inflation pressures.[14] Larger bags (greater than 500 mL) should probably not be used due to their inaccuracy when delivering 6-7 mL/kg tidal volumes. A main advantage of the self-inflating systems is their utility despite loss of fresh-gas flow. Certain bags may allow delivery of flow-by oxygen.[15]

Several difficulties complicate use of self-inflating bags for neonatal use, however. Most systems have preset (or adjustable) pop-off valves that allow a maximum pressure of 35 cm H_2O. On occasion, however, initial pressures of up to 40 cm H_2O are necessary to adequately expand the newborn lung.[16] Much of the bag's volume may empty through the valve without the operators being alerted to the presence of airway obstruction or noncompliant lungs. During bag-mask ventilation, leaks around the mask may be the only pressure relief required.[17] Finally, placement of the bulky reservoir bag at the airway in a small field impairs access to the patient and makes maintaining a seal more difficult.

Due to the anatomic considerations described, an oral airway is seldom necessary for neonatal resuscitation. However, airways of appropriate size should be available because nasal obstruction (eg, choanal atresia) or other difficulties may present themselves. Fortunately, bag-and-mask ventilation frequently may be successful despite inadequate gas movement, as inflation pressures may stimulate the baby to make her own inspiratory efforts (Head's paradoxical reflex).[18] One resuscitation system (the Samson) is designed to elicit this reflex yet is inappropriate (and perhaps dangerous) during prolonged resuscitation.[19] When the airway is difficult to manage and high inflation pressures are utilized, gas distention of the abdomen may be disasterous. The enlarging abdomen impairs ventilation via direct thoracic pressure, limitation of diaphragmatic movement, and distortion of chest geometry.

Volume-controlled resuscitators (VCR) are currently being evaluated. In one study with a small number of patients,[20] less variation in peak inspiratory pressure and less hyperventilation were noted, with mean airway pressure consistent with the standard resuscitator. Volume-controlled resuscitation is an important subject for future research.

Infant Masks. Two types of resuscitation masks are commonly employed in the delivery room. In 1960, Rendell-Baker and Soucek designed a mask to conform to infant anatomy, with very low dead space and an orifice for an adapter directly above the patient's nose (Fig. 3). This was the first pediat-

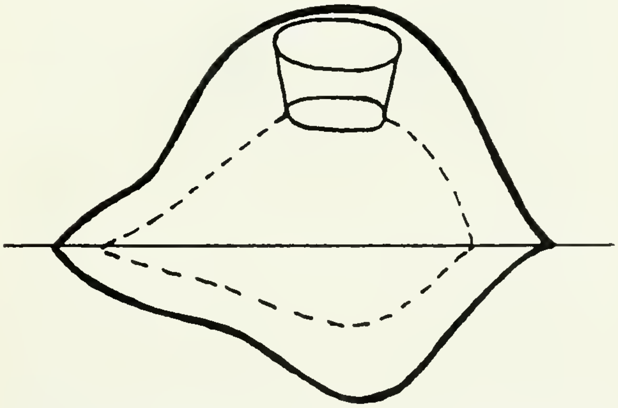

Fig. 3. Rendell-Baker-Soucek mask with dead space indicated (----).

ric mask fitted for a 22-mm adapter, which permitted attachment to standard (adult) breathing systems. Sizes range from 0 (premature) to 4 (age 4-8 y). The primary disadvantage of the Rendell-Baker-Soucek mask is the difficulty encountered in seating without substantial leak.[21]

The round mask in common use seats more easily and allows little leak, yet probably has greater dead space (although this has not been adequately studied) (Fig. 4). As with all masks, the proper size must be selected. With a mask that is too small, leaks are common and the nose may not be completely covered; when the mask is too large, the eyes may be damaged.

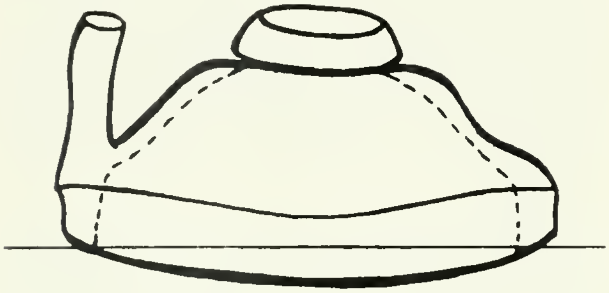

Fig. 4. Round mask with dead space indicated (----).

Endotracheal Tubes. In 1945, Cole cemented a 3.5-mm-ETT tip to a 5.5-mm ETT in order to prevent endobronchial intubation in small patients.[22] The site of connection, or 'shoulder,' of the tube stopped at the cricoid with the tip resting safely above the carina. Although this tube was once popular, it is no longer recommended by the American Academy of Pediatrics or the American Heart Association.[23] Disadvantages of the Cole tube include (1) difficulty in passing a suction catheter to the tip, (2) difficulty in manipulation and visualization during intubation (due to its larger diameter), and, most importantly, (3) higher incidence of laryngeal damage with chronic use.[24] Additionally, the Cole tube may provide a false sense of security and, thus, slow recognition of endobronchial intubation.

Conventional ETTs without taper (of which there are several manufacturers and construction materials) have both centimeter and vocal cord markers (Fig. 5). Many are designed so that placement of the double lines at the cords results in proper tip positioning. Additionally, the tip bevel allows smooth passage through the nares and a side hole allows ventilation when the tip is obstructed or placed slightly into a main-stem bronchus.

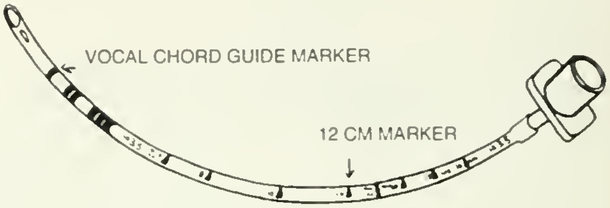

Fig. 5. Endotracheal tube with markings.

A number of approaches have been used to estimate the correct tube size for the infant. One of the more popular ways is to use the patient gestational age[25] (Table 4). Perhaps the easiest method for determination of the correct ETT size in the delivery room is to think of small (2.5 mm), medium (3.0 mm), and large (3.5 mm). A premature infant requires a 2.5-mm tube and a full-term a 3.5. Other formulas have been developed to estimate tube size. One is the ID = 16 + patient age in years/4.[26] Others have used height and age to predict size[27] or body-surface area and size.[28] The tube should be large enough to prevent excessive leak. In up to 8% of premature infants, the tracheal diameter is smaller than the cricoid diameter.[29] The average distance from the glottis to the carina in newborns is 5.7 cm, and the distance from the lip to the proper position of the tube tip is approximately 7 cm at 1 kg, 8 cm at 2 kg, and 9 cm at 3 kg.[30] The

Table 4. Suggested Endotracheal Tube Size*

Age	Internal Diameter (mm)
Premature infant	2.5-3.0
Newborn	3.0
Newborn-6 months	3.5
6-12 months	3.5-4.0
12 months-2 years	4.0-4.5
3-4 years	4.5-5.0
5-6 years	5.0-5.5
7-8 years	5.5-6.0
9-10 years	6.0-6.5
11-12 years	6.5-7.0
13-14 years	7.0-7.5

*Adapted from Reference 23.

average tube movement from full flexion of the head to full extension is 14-17 mm.[31] This movement should be considered during x-ray interpretation of tube placement.

Recently, a new device, the Trach-Mate (McCormick Laboratories, North Chelmsford MA), has been introduced to facilitate rapid verification of ETT positioning.[32] This device makes use of a magnetically detectable ring embedded in the wall of the endotracheal tube, which can be detected by a battery-powered locater placed externally at the sternal notch. However, auscultation and chest x-ray remain the standards of position assessment.

Laryngoscopes. Like suction, short-handled laryngoscopes with bright lights and both Miller 0 and Miller 1 blades must be available in every delivery room. We prefer straight blades over curved blades for approach to the more cephalad, anteriorly situated neonatal larynx.

Airway Management in the Delivery Room

Management of the newborn airway is the most critical element of neonatal resuscitation. Not uncommonly, establishment of airway patency and delivery of several breaths is all that is required to transform a dying infant into a healthy, vigorous one. As the newborn lungs are expanded and pulmonary vascular resistance is decreased, the transition from fetal to normal circulation is made. Failure to make this transition is incompatible with life.

Guidelines from the American Heart Association and the American Academy of Pediatrics[23] recommend that copious secretions be suctioned from the newborn's mouth and nose with a bulb syringe. This, in addition to clearing the airway, may provide enough stimulation to induce the baby to breathe effectively.

Suctioning. The mouth is always suctioned first, and deep suctioning should be avoided lest vagal stimulation produce bradycardia. An exception to this arises in the case of meconium staining. Thin, watery amniotic fluid requires no special treatment. Thick, 'pea soup,' particulate meconium requires immediate suctioning as the head is delivered (ie, before the first breath). The density of meconium mandates the type of suctioning required: bulb syringe (Fig. 6), DeLee (Fig. 7), or direct via the endotracheal tube (Fig. 8). In any case, endotracheal suction pressure should not exceed 100 torr.

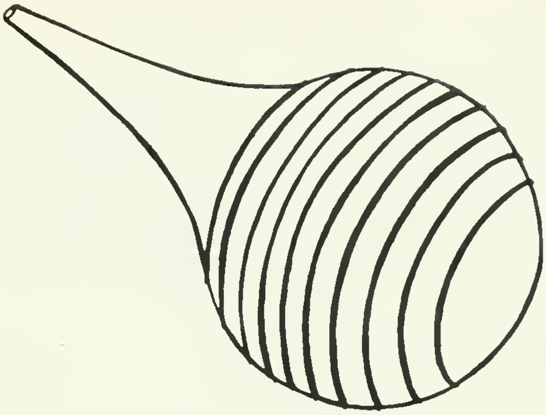

Fig. 6. Delivery room bulb syringe.

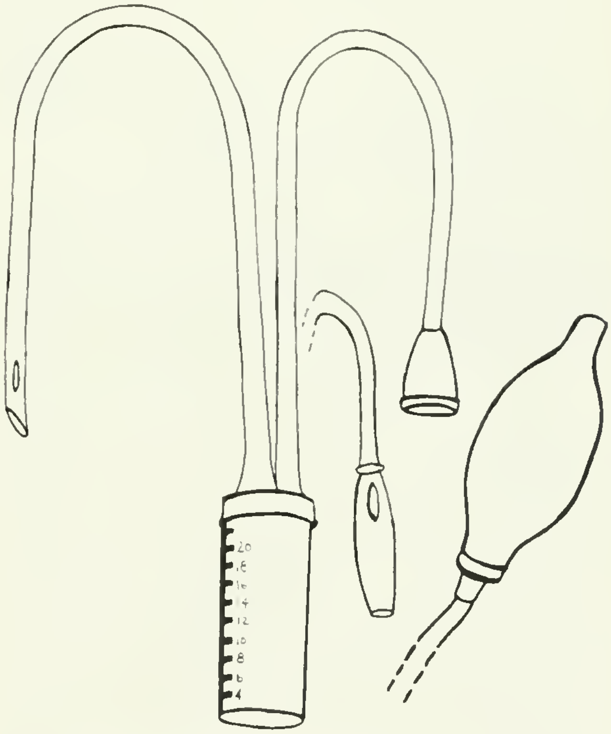

Fig. 7. DeLee suction and its variations.

Choanal Atresia. Congenital obstruction of one or both choanae occurs in approximately 1 in 8,000 live births. Obstructions may be bony or membranous in nature. As neonates are nearly obligate nose breathers until 3-6 months of age under most circumstances,[33] such obstructions are a serious matter. Presentation is usually that of a cyanotic

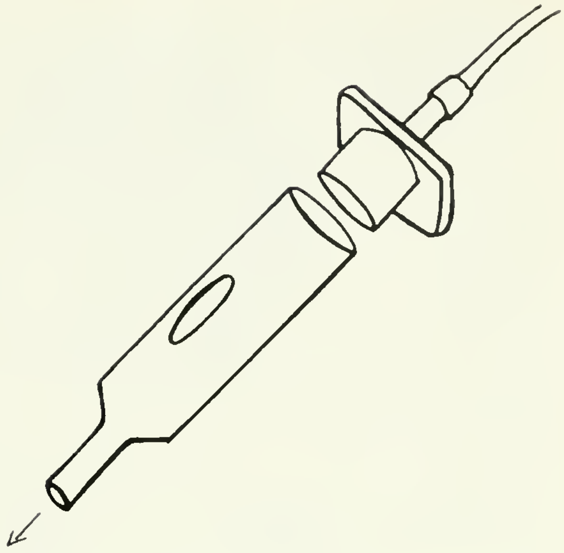

Fig. 8. Adapter for direct suctioning to endotracheal tube.

infant who pinkens with crying. The diagnosis is confirmed when it is found that a soft suction catheter cannot be passed through the nares. An oral airway should be placed immediately.

Meconium Aspiration Syndrome. Meconium is present in the amniotic fluid in 10-15% of all births and should alert the delivery team to possible complications. The passage of meconium (a mixture of digested epithelial cells, bile, and gastrointestinal mucus) is generally regarded as abnormal and can accompany in-utero stresses such as hypoxemia.[34-36] Meconium aspirated into the airway can act both as an irritant and an obstruction. If meconium-stained fluid is observed upon rupture of membranes, preparation must be made to suction the oro- and nasopharynx as soon as the head appears on the perineum.[34,37,38] When thick meconium is present, the infant should be intubated and suctioned at the first sign of airway compromise. Positive pressure ventilation should be avoided until the airway has been cleared so that particulate material is not forced into the distal airways.[39]

Approximately 5% of meconium-stained infants go on to develop meconium aspiration syndrome (MAS).[37] MAS consists of severe, diffuse pneumonitis and typically includes varying degrees of pulmonary hypertension with shunting and hypoxemia. Common complications include pneumothorax, pneumomediastinum, and, rarely, pneumopericardium (secondary to the high inflation pressures required for ventilation and to air trapping with alveolar overdistention distal to meconium obstruction). Radiographs of the chest are notable for the presence of patchy densities interspersed with radiolucence.[35] Although many infants respond to conventional respiratory support and mechanical ventilation, high frequency ventilation[40] or extracorporeal membrane oxygenation[41] (ECMO) is sometimes necessary for rescue.

Congenital Diaphragmatic Hernia (CDH). In-utero herniation of abdominal contents via defects in the right or left (70%) hemidiaphragms occurs in approximately 1 in 4,000 live births.[42] Such herniation is accompanied by severe pulmonary hypoplasia ipsilaterally and variable degrees of hypoplasia contralaterally. Pulmonary hypoplasia is frequently compounded by severe pulmonary hypertension with shunting that presents shortly after birth. Frequently, severe decompensation occurs after a period of initial stability (the 'honeymoon' period).

Diagnosis is suggested in the delivery room by respiratory distress and a scaphoid abdomen. Auscultation reveals absent breath sounds on the side of the defect and, perhaps, displacement of the heart tones. Diagnosis is confirmed by a chest x-ray demonstrating abdominal organs in the thorax and, often, a mediastinal shift. When CDH is suspected, bag-mask ventilation should cease immediately and the infant should be intubated. Mask ventilation will lead to gastric distention with air and further impingement upon thoracic organs. For similar reasons, the stomach should be decompressed with a gastric tube. A frequent complication of respiratory support in these patients, who typically require high inflation pressures, is contralateral pneumothorax. When this occurs, the results are rapid and disastrous for obvious reasons. Because of this risk, many practitioners will place chest tubes prophylactically in patients before transport to referral centers for definitive management.

The cause of CDH is still unknown. One possibility is that the defect in the diaphragm causes compression of the lung resulting in hypoplasia.[43] Another belief is that the lung hypoplasia comes first and leads to incomplete development of the diaphragm.[44] Most agree that the embryologic insult occurs during the eighth week of gestation, causing failure of closure of the pleuroperitoneal canal, most commonly at the foramen of Bochdalek.

The survival rate remains approximately 50% if the infant is symptomatic within the first 6 hours of life.[45,46] Predictors for mortality have been proposed utilizing pH, P_{aCO_2}, $P_{(A-a)O_2}$,[46-49] pulmonary angiography,[43] P_{aCO_2} versus ventilation index (VI) (ventilation rate × mean airway pressure),[50] and best postductal P_{O_2}.[51] All of these have been criticized during the ECMO era. A recently described predictor of poor outcome is the oxygenation ventilation index, or OVI (P_{O_2} divided by the ventilation index, VI).[52]

Subsequent management is directed by the severity of pulmonary disease. Mildly affected neonates may undergo surgical repair after a brief period of observation and may tolerate the procedure well.[53] Patients with severe disease may be difficult or impossible to stabilize with conventional treatment. In our institution, these infants are offered ECMO for rescue. Surgical repair may then take place later—on or after separation. It is hoped that new approaches such as antenatal intervention,[54] ECMO, lung transplantation, and delayed surgery will increase the survival rate in this group.

Surfactant Administration. In premature infants with early distress syndrome, surfactant may be given in the delivery room. In one study,[55] two controlled trials were conducted.

1. Prophylactic trial: 5 mL/kg of surfactant through the ETT at birth to neonates weighing 700-1,350 g.

2. Rescue trial: 5 mL/kg given 4 to 24 hours after birth to neonates greater than 650 g with a diagnosis of hyaline membrane disease who required ventilatory support.

Both trials decreased F_{IO_2} and mean airway pressure requirements. In the prophylactic trial, 30% of the infants who received surfactant probably did not need it. In our institution, because of this and the success of the rescue trial, we seldom use surfactant in the delivery room.

Pediatric Airway Management

As the child grows to adulthood, differences in airway anatomy gradually disappear. The epiglottis, for example, descends from its contact with the soft palate in the newborn period to its adult level (at approximately C3, the third cervical vertebra) by the fourth year of life. Similarly, the glottis has reached the C4-5 level by this time.[56] The cricoid cartilage grows to a diameter greater than the glottic opening during the early school years and descends to adult levels later after pubertal elongation of laryngeal structures. However, during the pre- and early school years, the child remains vulnerable to airway compromise and apparently simple respiratory illnesses may rapidly evolve into life-threatening crises. The clinician must be familiar with pediatric equipment and pathology to provide the prompt response required in such emergencies.

Pediatric Equipment

As sizes and anatomic relationships in the airway change, appropriately sized and designed equipment must be employed. It is prudent to use age and/or weight in kilograms to estimate the size of equipment necessary and then set out equipment of that size and a size smaller and one larger. This takes into account individual variations and prepares the clinician for the unexpected.

Endotracheal Tubes. A variety of systems have been developed for estimating the appropriate ETT size. In younger children, as in infants, the general goal is to insert a tube that passes through the glottis atraumatically and results in an appropriate seal (< 20-25 cm H_2O) at the cricoid ring. In older children (usually age ≥ 8 years), a cuffed endotracheal tube is permissable with the seal provided by gentle inflation. Although the seasoned practitioner may accurately select ETT size through 'eyeball and experience' or by approximation of the size of the child's fifth finger, a variety of formulas for tube selection have been suggested. In our institution, the most popular of these has been (age in years + 18)/4.[57]

Appropriate insertion distance has also been the subject of estimation formulas. Although there is no substitute for direct visualization (radiographically or via laryngoscopy), occasionally this is impractical. When position is critical, some practitioners manipulate the tube to intentionally produce an endobronchial intubation and then auscultate carefully as the tube is slowly withdrawn. Proper position is then taken to be 1-2 cm beyond the point at which breath sounds return. The taping position of oral tubes may be rapidly estimated (with variable accuracy) through formulas such as: 3 × tube ID; age in years/2 + 12;[58] and weight in kg/5 + 12.[59] Finally, when frequent head repositioning is anticipated, auscultation should be performed in neck flexion and extension. Flexion shortens the mouth-to-carina distance, thus displacing the ETT tip downward and increasing the danger of endobronchial intubation. Conversely, extension increases this distance, promoting inadvertent extubation.

Occasionally, airway pathology substantially limits the allowable tube diameter. In such cases, smaller than usual tubes should be readily available in a wide variety of sizes. Additionally, the need for extra-long tubes should be anticipated should standard smaller diameter tubes be too short.

Laryngoscopes. In young children, straight-blade laryngoscopes are generally preferred, yet curved blades of appropriate size are available as well. Addition of curvature to the body of a blade can make visualization of an anteriorly situated glottis signficantly more difficult. The broad blade and flange, however, can be quite helpful in tongue displacement and actually improves visibility in some patients.

The most popular straight blades are the Miller, Wisconsin, and Wis-Hipple varieties. Dr Robert A Miller of San Antonio, Texas, designed his straight-blade variation with a reduced flange size to minimize trauma and a curved tip to improve lifting of the epiglottis.[60] Miller-type blades range in size from 0 (for prematures) to 4 (for large adults). Size 2 is appropriate for children of approximately 6-12 years. The Wisconsin blade features a flattened tip and a larger flange that widens further in the distal portions. The Wis-Hipple blade, a modification of the Wisconsin blade for use in infants, employs a circular flange in addition to the straight spatula. The Wis-Hipple 1.5 is often useful in children 2 to 6 years old.

Macintosh introduced what is now the most used curved blade in 1941.[60] It is designed so that the tip may be placed into the vallecula, the blade lifted, and the epiglottis indirectly raised to reveal the cords. The wide flange and broad spatula facilitate displacement of the tongue. In most patients, this large blade provides increased visibility and room to maneuver, yet in very small patients it may be too bulky and obstructive. Like Miller blades, Macintosh blades range in size from 0 (newborn) to 4 (large adult). Most children may be intubated using a Size 2 blade and adults a Size 3.

A variety of other laryngoscopes may be employed in unusual circumstances. The Oxyscope (a straight-blade with a port incorporated for continuous insufflation of oxygen) has been advocated to prevent hypoxemia.[61] A 33% decrease in P_{aO_2} during intubation has been reported without supplemental oxygen, with only a 7% decrease with supplemental oxygen.[62] A pediatric version of the Bullard laryngoscope provides an indirect view of laryngeal structures via mirrors and fiberoptics.[63] It has been suggested for use in cases of severe micrognathia, macroglossia, and other disorders.

Resuscitation Equipment. Nondisposable adult resuscitation bags have been studied over the years.[64-68] Manufacturers reduce the size (volume) of the adult bags to meet the needs of the pediatric patient, but the valving system remains the same. Pediatric disposable resuscitation bags more recently have been evaluated.[69,70] Barnes and McGarry[70] found that the four pediatric disposable resuscitation bags tested failed to meet all aspects of Standard F-920-85 of the American Society for Testing and Materials.[71] Emergency Care Research Institute (ECRI)[69] found four pediatric resuscitation bags acceptable, but two did not have a pop-off valve. Respiratory care practitioners should be aware of the ASTM F-920-85 standard and the performance of the resuscitation bag they are currently using.

Pediatric Airway Emergencies

Acute airway compromise in pediatric patients arises most commonly either from infectious swelling of airway structures or inadvertent aspiration of a foreign body. In his interesting historical review, Stool notes that prior to 1900, the term "croup" was equated with laryngeal diphtheria.[72] Technical complications of pediatric tracheostomy prevented adequate treatment of diphtherial airway obstruction until the work of O'Dwyer in the late 1800s. O'Dwyer, a New York physician, developed an intubation system consisting of stainless steel tubes and an introducer device. A team was required for restraint of the child, yet intubation eventually led to relief and survival. As surgical technique improved, controversy developed regarding tracheostomy versus intubation (the steel tubes being responsible for cases of severe tracheal and laryngeal damage). Fortunately, diphtheria immunization lessened the need to resolve the dispute.

Laryngotracheobronchitis. Today the term croup is typically applied to viral laryngotracheobronchitis. Parainfluenza Type 2, respiratory syncytial virus, adenovirus, or other upper-respiratory pathogens are probably responsible yet rarely identified.[73,74] The presentation is typically that of barking cough progressing to stridor in a young child (6 months to 2 years) in the setting of an otherwise benign upper-respiratory illness.[75,76] Varying degrees of respiratory distress may be present (including respiratory fatigue with failure) as well as low-grade fever.[77] A Spring-Fall seasonal predominance has been observed.

Pathologic airway changes include inflammation and edema of the laryngeal, tracheal, and bronchial mucosa. Circumferential edema results in substantial reductions in airway cross-sectional area, with an increase in turbulent flow and resistance. Radiographic findings classically include a normal epiglottis (on lateral view) and, on posteroanterior films, subglottic narrowing (the so-called steeple sign).[78]

In most cases, croup resolves with conservative therapy: humidification of inspired gases, supplemental oxygen, and systemic hydration.[76,78,79] In more severe cases, aerosolized racemic epinephrine is necessary to decrease edema and improve airflow. These patients require hospital admission should more severe airway compromise occur once the medication's effects have dissipated. Occasionally, severe cases (1%) require endotracheal intubation to stent the airway until edema abates. When intubation is required, it should be attempted in a controlled setting by experienced personnel.[79] An ETT one size smaller than estimated by conventional methods should be used.

Postintubation care should include adequate sedation (to avoid tube motion and additional traumatic edema) and respiratory support. Mechanical ventilation is occasionally necessary in heavily sedated or debilitated patients; however, our experience has been that the majority do well in mist tents. If mechanical ventilation is not required, attachments to the ETT (such as T-pieces and CPAP devices) are best avoided as they are inclined to put tension upon (and move) the ETT. Patients with croup can usually be extubated within 48-72 hours. In our unit, we find that extubation may be safely performed when an audible air leak is present around the ETT at 20-30 cm H_2O (as measured by in-line manometry during manual chest inflation).

Over the years, steroid treatment for croup has waxed and waned in popularity. Although still controversial, there is increasing evidence that administration of steroids may diminish both the duration and severity of croup episodes.[80-82] Dexamethasone 0.6-1 mg/kg has been recommended.[83]

Epiglottitis. Epiglottitis is a rapidly progressive, life-threatening, bacterial infection of supraglottic structures. In contrast to croup, it is always a true medical emergency requiring intubation.[75,84] Cardinal features include sore throat, dysphagia, drooling, muffled voice, respiratory distress in an anxious, toxic-appearing child.[73] *Haemophilus influenzae* Type B is the organism responsible in nearly all cases and may be readily cultured from the blood.[73,76,84] Winter-Spring seasonal predominance has been seen, and all age groups have been affected. One series reported 24% of cases under age 2, 20% over age 4, and 55% between 2 and 4.[85]

When epiglottitis is suspected, an anesthesiologist and otolaryngologist should be summoned immediately. Care should be taken to avoid disturbing the child, and supplemental oxygen should be provided. If the diagnosis is uncertain and x-ray confirmation is required, the child should be accompanied to the radiology suite by both physicians with equipment necessary for intubation and tracheostomy. Confirmatory radiographic findings on lateral neck films include thickening or smudging of the epiglottic shadow ("thumb sign"),[78] obliteration of the vallecula and pyriform sinuses, and dilation of the hypopharynx. Care must be taken to assure a true lateral film (locating the hyoid bone or auditory canal can confirm this). In our institution, once the diagnosis is made, the patient is transported (accompanied by a parent) to the operating room where anesthesia is cautiously induced by inhalation. Once anesthetized and with spontaneous respiration maintained, the patient is intubated. Swab cultures of the supraglottic regions are then sent and antibiotics begun. Further management is similar to that of croup.

Extubation is usually possible 36-60 hours after initiation of antibiotics.[86] Unlike croup, air leaks in a patient with epiglottitis may be misleading. Although air may leak upwards with positive pressure, negative pressure during normal respiration may still cause an inflammed and enlarged epiglottis to collapse and obstruct the airway. When uncertainty exists, direct visualization prior to extubation can exclude the presence of residual swelling.

Airway Obstruction in Pediatric Patients

Partial or total airway obstruction may occur in a variety of forms in pediatric patients and is responsible for significant morbidity and mortality. As discussed, the young child is predisposed to respiratory compromise. Additionally, small children are prone to oral indiscriminations resulting in foreign-body aspiration. Respiratory dysfunction is a primary and common cause of cardiac arrest in children.[87]

Signs and symptoms of obstruction include nasal flaring, retractions, tachypnea, stridor, wheezing, rhonchi, and prolonged inspiratory or expiratory phases. Generally, normal breathing is inaudible and thus concern is raised when noises appear.

Nasal Obstruction. At the nasal level, obstruction most commonly occurs secondary to inflammation of mucous membranes due to viral upper-respiratory infections. As noted above, this is trivial in adults and older children yet can be a serious problem in the infant. Sinusitis may produce thick, copious secretions that may also block the airway. Other common causes of obstruction at this level include adenoidal hypertrophy, septal deviation, polyps, and, rarely, neoplasm. Often, snoring is the sound first associated with obstruction at this level.[4] Obstruction may also occur at the oropharyngeal level. While tonsillar hypertrophy (sometimes leading to sleep apnea, CO_2 retention, and cor pulmonale) is probably the most common obstruction here, macroglossia, micrognathia, retropharyngeal abscesses, and cysts may also be present. Often a snore or gurgling respiratory sounds are associated.

Laryngeal Obstruction. Laryngeal obstructions are also common.[2] Laryngomalacia, cysts, webs, and stenoses may be congenital or the result of prolonged intubation. Vocal-cord paralysis (or dysfunction), vesicular malformations, granulomas, papillomas, and neoplasms also occur. Infection and foreign bodies are very common, as discussed below. Obstruction around the glottis results in stridor and hoarseness. Supraglottic lesions produce muffling. Tracheal obstructions may form (including stenoses, vascular rings and slings, malacia, cysts, webs, and hemangiomas). When tracheal compression is extrinsic and associated with esophageal compression, feeding difficulties may be present. Fixed obstruction may produce both inspiratory and expiratory (biphasic) stridor. Extrathoracic variable obstruction produces primarily inspiratory stridor, and intrathoracic variable obstruction produces primarily expiratory stridor. The most common intrathoracic airway obstruction (and, indeed, the most common chronic illness) in childhood is asthma. Small-airway obstruction becomes manifest as inspiratory and expiratory wheezing, with the expiratory component greater.

Foreign-Body Aspiration. Airway obstruction due to aspiration of foreign bodies is most common in patients less than 6 years of age and is responsible for over 3,000 deaths annually in the U.S. The majority (85%) of these occur in patients under 3 years, making it the most common cause of death in this age group. A review of childhood asphyxiation (0-9 years) in the United States during a 3-year period revealed 60% male predominance, 94% under 5 years of age, and 61% under 1 year of age.[88] Although nuts, pins, coins, and pebbles are the conventionally described aspirate,[73] hot dogs have probably become more common. In younger children, balloons may be particularly hazardous.

In some series, the right lung is more likely to be affected than the left, yet in others (particularly in the youngest children) rates are equal.[89,90] In 75% of the cases, the object lodges in the main-stem or lobar bronchus, and in 20% the object lodges in the upper airway. Mechanisms of airway obstruction include (1) the check-valve obstruction (air is inhaled but only partially exhaled, resulting in overinflation), (2) the stop-valve obstruction (air cannot enter, resulting in atelectasis), (3) the ball-valve obstruction (foreign body dislodges during expiration and reimplants during inspiration), and (4) the bypass-valve obstruction (the lumen is only partially obstructed, resulting in decreased aeration and opacity).[91]

Signs of foreign-body aspiration may be subtle and include only coughing or wheezing of a chronic nature rather than the dramatic choking, dyspnea, and cyanosis more commonly recognized. Physical findings include decreased air entry or wheezing (the latter often bilateral), tachypnea, rales, and, in about one third of patients, fever. Radiographically, the object may be visible or infiltrates may be seen (with or without emphysema), yet often the films are normal. Other radiographic findings present less frequently include pneumothorax, pneumonia, and bronchiectasis. Appropriate radiographic evaluation may include inspiratory and expiratory films or lateral decubitus films; however, in our experience fluoroscopy is more reliable.

In addition to asphyxia, complications seen with foreign-body aspiration include pneumonia, pulmonary edema, atelectasis, bronchitis and bronchiectasis, pulmonary abscess, bronchopulmonary fistula, and bronchospasm.

The treatment of foreign-body aspiration usually requires rigid bronchoscopy in the operating room under general anesthesia. In an emergency, out-of-hospital treatment includes back blows/chest thrusts on infants and the Heimlich maneuver on children. A more detailed description is available in the Pediatric Advanced Life Support (PALS) series.[92]

Securing the Artificial Airway

The unexpected loss of an artificial airway in an infant or child may, at best, end in a successful extubation or, at worst, result in accidental death. Accidental extubation (AE) has been associated with an increased risk for subglottic stenosis[93] and mortality.[94-96] The incidence of AE has been reported to be anywhere from 1% to 46%[97,98] in neonates and children. Most authors fail to specifically define AE; however, common to all studies is the unexpected loss of an ETT, whether this be complete displacement or more subtle displacement as evidenced by sudden increased phonation. No distinct advantage of nasal over oral intubation has been demonstrated with respect to AE; however, at least one author feels the nasal route may provide greater stability.[93]

Numerous factors have been implicated in contributing to AE. Use of restraints, taping methods,

Table 5. Taping Methods for Securing Endotracheal Tubes*

Author	Securing Method
Benjamin et al[100]	White cloth adhesive tape
Brown[98]	Cloth/elastic/pink tape
Budd[102]	Logan Bow and adhesive tape
Wood & Sherman[101]	Adhesive tape
Nugent et al[103]	Elastic/pink tape and sutures through ETT
Kerr[104]	Facial/ETT strap
Molho & Lieberman[105]	Modified latex nasopharyngeal airway, cotton strings, and suture
Nieves[106]	Safety pin or umbilical clamp or tape
Richards[107]	Double-stick disk and adhesive tape
Robson & Tompkins[108]	Vapor-permeable dressing, silk/stretch tape
Schuller & Birck[109]	Logan Bow and tape
Striker et al[94]	ETT wall sutures and tape
Valley & Norfleet[110]	Wire stabilizing device, headstrap, and tape
Rivera & Tibballs[111]	Knotted string, tape, and elastoplast

*Adapted, with permission, from Reference 111.

use of sedation, chest physiotherapy, suctioning procedures, and length and size of ETT are some of the variables cited in the incidence of AE.[98] Assessing the influence of each of these variables is a nearly impossible task because much of the literature on AE has focused on reporting incidence rather than individual causes. In a study by Scott et al,[99] four variables were found to be predictive of accidental extubation: younger age, many secretions, ETT slippage, and higher level of consciousness. Brown[98] has found a specific taping method to be important in minimizing accidental extubations; yet even with the preferred method and head restraint, the incidence of AE was 23% in their report. The diversity among ETT stabilization methods is shown in Table 5.[94,98,100-111] Several types of tape and various specialized headstraps and sutures are some of the materials used by clinicians to secure ETTs. In our institution, the previously reported AE incidence has been a relatively low 3% with the use of white cloth adhesive tape in a Y configuration.[100] The variety of taping methods used reflects the challenge that securing an infant or child's ETT can be. The importance of a stable, secure artificial airway is obvious, and the perfect taping method is not.

In Conclusion

The anatomy, physiology, equipment, and strategies of pediatric airway emergencies differ greatly from those of the adult. There are no standards for training in the delivery room. We recommend that persons gain experience in a Level-III nursery before assuming responsibility for resuscitation in the delivery room. Pediatric Advanced Life Support and Neonatal Advanced Life Support courses are definitely helpful, but experience is necessary.

ACKNOWLEDGMENTS

We are grateful to Catherine Ferrarini and Patrice Benjamin for their asssistance in the preparation of this manuscript.

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Thompson Discussion

Jeffs: When you are doing oral suction for meconium, do you go ahead and intubate and suction as well then?

Thompson: Oral suctioning with the bulb syringe? The important thing is not to go past the tongue because you'll get a vagal stimulation and bradycardia; so, that is really just for the external nares and the oral cavity. For anything deeper than that we use the DeLee suction.

Jeffs: But, is it your protocol that once you see signs of meconium that you automatically intubate them to check?

Thompson: No, actually it depends on what comes out in the amniotic fluid after the membranes are broken. If it's just meconium stained, then as soon as the head is delivered we use a bulb syringe and look to see if it's watery. If it's watery, we don't do anything more. We treat the infant like a normal newborn. If there's any particulate meconium, then as soon as the head is delivered, we deep suction. When the baby's delivered then it depends, but usually we intubate.

Jeffs: My second question has to do with surfactant use. When surfactant administration was first being studied, they advocated that you shoot the surfactant down before you give a breath to the baby—just 'tube 'em' and shoot it. But you're saying to wait 2 hours?

Thompson: I'm saying that under that protocol, approximately 30% of the patients received a dose of surfactant that they really didn't need in comparison to control groups. Both prophylactic and rescue groups ended up with a decrease in ventilatory support requirements and a decrease in F_{IO_2} requirements.[1] Because both protocols worked equally well, to avoid giving surfactant to the 30% who really don't need it, we very seldom have it ready in the delivery room.

1. Phibbs RH, Ballard RA, Clements JA, Heilbron DC, Phibbs CS, Schlueter MA, et al. Initial clinical trial of EXOSURF, a protein-free synthetic surfactant, for the prophylaxis and early treatment of hyaline membrane disease. Pediatrics 1991;88(1):1-9.

Jeffs: What is the protocol you follow?

Thompson: Just the rescue protocol.[1] Once patients are observed to be having trouble with respirations, they are intubated and given surfactant at 5 mL/kg. Then they're eligible 12 hours after that to get a second dose. That's under the rescue protocol.

1. Phibbs RH, Ballard RA, Clements JA, Heilbron DC, Phibbs CS, Schlueter MA, et al. Initial clinical trial of EXOSURF, a protein-free synthetic surfactant, for the prophylaxis and early treatment of hyaline membrane disease. Pediatrics 1991;88(1):1-9.

Reines: Who does your intubations—pediatric and neonatal—the respiratory care practitioner or the physician? And a totally unrelated question: Who in your unit gets 'trached?' When? And if you don't trach them, what's your incidence of problems?

Thompson: Who does intubations? It's physicians in our institution. We're just like Bob's (Kacmarek) institution. We have a million people around, a million physicians to train. It's the physicians who intubate. In the delivery room, depending on what's going on, we get called in to do the resuscitation, not necessarily the intubation. If we know that the pH of the scalp blood sample was bad, and that we're getting a sick baby, then the therapist will do bag-mask ventilation. About tracheostomies—we go months and months and months with intubation and without traching patients. We trach patients mainly because we know that they're going to need positive pressure ventilation for long periods of time (home ventilation) or because of some anatomic problem or need for laser therapy. A lot of patients who get laser therapy are sent home with metal trachs for a long period of time.

Barnes: I have a question about intubation during newborn resuscitation. In one series of 10 resuscitations at birth reported by Milner et al,[1] none of the infants ventilated with a Laerdal self-inflating bag-valve-mask resuscitator could be adequately ventilated, assuming that to be a tidal volume of 2.2 mL/kg body weight. However, 5 of 9 intubated babies exceeded this level. Would you comment on the need to intubate infants at birth who do not start to breathe within 2 minutes?

1. Milner AD, Vyas H, Hopkin IE. Efficacy of facemask resuscitation at birth. Br Med J Clin Res 1984;289:1563-1565.

Thompson: Are you saying that they intubated 100% of the patients?

Barnes: They could not establish an adequate tidal volume at birth for 10 babies who were not intubated. In 9 babies who were intubated, only 5 were able to be adequately ventilated.

Thompson: Again, what you're asking is, What is the best way to establish FRC (functional residual capacity) in a newborn? The best way to establish FRC in a newborn is to let the baby breathe spontaneously—not to intubate him. Usually, it takes just a couple of breaths with the bag to elicit spontaneous respiration. Once we go to intubation, then you're right; you may need high pressures to establish FRC.

Barnes: You're using what you call the Mapelson Type-F bag. I know that you like a lot of things about that bag, but as you said it's difficult to use. Can you recommend what level of staff should be taught to use that bag, and is it a bag that the average delivery room nurse can use effectively?

Thompson: If you don't use the bag every day, then I would say, "Don't use the bag." It does take practice. It certainly takes months to train. We have a 3-month orientation for new therapists, and it takes that long for someone to feel comfortable using that bag. Every one of my staff is trained in it because that's all we use. Even on ventilated pediatric patients, all we use is the Mapelson F. We don't use self-inflating bags. We bring self-inflating bags on transport, in case we lose gas, but for the most part we use all non-self-inflating bags. Once you get used to them, then you can't imagine going back to a self-inflating bag because of all the dangers of high inflation pressures and long inspiratory times and things like that. They're always used with a manometer.

Rodriguez: What do you think about using the NRPR (non-rebreathing pressure-relieving) elbow for bag-mask ventilation?

Thompson: Is that the Keat's or the Norman elbow?

Rodriguez: I think it was developed by Dr Robert Husted and is now distributed by Puritan-Bennett.

Thompson: Oh, is that the one that they thumb?

Rodriguez: No.

Thompson: I'm unfamiliar with that. What's coming around, though, is the volume-oriented resuscitation bags, the so-called VCRs, where you get a more consistent peak inspiratory pressure, less chance of hyperventilation with the volume-controlled resuscitators, but I'm not familiar with the device. I've not used it.

Mathews: What are you guys using to measure the effect of treatment or therapy in these upper-airway obstructions? The reason I ask is that there's been an interesting paper by Sivan, Becker, and Newth[1] that talks about using a respiratory plethysmograph to objectively measure phase angle between rib-cage and abdominal movements in children, and they're claiming that it's very selective in this area.

1. Sivan Y, Becker TW, Newth CJ. Thoracoabdominal asychrony in acute upper airway obstruction in small children. Am Rev Respir Dis 1990;142(3):540-544.

Thompson: I think that's the Respitrace you're referring to. We don't use the Respitrace. We use a lot of flow-volume loops and pressure-volume curves that Bob (Kacmarek) alluded to with the Hamilton and the VenTrak.

Mathews: You take them directly off the ventilator?

Thompson: Off the ventilator. We also have a lot of luck with the little NVM monitor, the neonatal volume monitor. It hooks directly into the VenTrak; so, you don't need to use a very large pneumotach, just the little hot-wire anemometer from the Bear neonatal volume monitor.

Barnes: There's a study by Goldstein in RESPIRATORY CARE that showed that if you don't use a manometer with bag-valve ventilation that the peak inspiratory pressure may be 6.5 cm higher.[1] Is that your experience, to use manometers all the time when you do bag-valve ventilation?

1. Goldstein B, Catlin EA, Vetere JM, Arguin LJ. The role of in-line manometers in minimizing peak and mean airway pressure during the hand-regulated ventilation of newborn infants. Respir Care 1989;34:23-27.

Thompson: Yes, that is our experience. A lot of the neonatal bags come with fixed leaks. When you squeeze them, that fixed leak creates a lot of turbulence, and your inspiratory time will be much shorter at a very high pressure. Even though you can generate a very high pressure at the airway, it's really not transmitted distally. So, a lot of those bags may generate 80 cm H_2O at the airway but still only 10 or 15 cm H_2O distally—especially bags like the Cardiff or Penlon.

Barnes: How important is it to maintain PEEP levels during 'bagging' with neonates? Goldstein's study showed that with a manometer you couldn't maintain PEEP very well, even though you could control peak inspiratory pressure. How do you control that? Or is it not a problem?

Thompson: Cerebrally it's a problem. We think that if a patient on a ventilator needs 10 cm H_2O PEEP, then during bagging he needs 10 cm H_2O PEEP. So, I don't know, Tom (Barnes). I would say that every time someone picks up a bag, the respiratory rate goes up much higher than the set rate on the ventilator, so that certainly any mean airway pressure that we lose when we lose PEEP is made up by the rate at which one 'bags.'

Near-Drowning

Martin J Nemiroff MD

River Dragon

River dragons lurk
in every Chinese river,
but they've swum across the sea:
I know they lurk here too.

In spring river dragons suck
water from the fields and
puddles from the streets.

River dragons wait
for boys on shore to slip
within the water's reach.

River dragons prefer
the young who are harder
to subdue.

River dragons grow.
They fatten in the spring
on water and young boys.

Leslie Y Lin

Introduction

Since 1975, there has been a renewed interest in resuscitation following prolonged submersion in water. A re-examination of techniques and protocols for rescue and resuscitation began after the publication of reports of complete neurologic recovery after 38-minute[1-4] and 40-minute[5] submersions. The distinction became less clear between drowning (death by asphyxia after submersion) and near-drowning (at least temporary survival after submersion). Further confusion appeared when terms such as immersion syndrome, immersion pneumonitis, and secondary drowning were used.

In current usage, the term drowning means death during the first 24 hours after a submersion. Near-drowning, a term first used by Modell in a 1971 book,[6] now is used to describe the survival for at least 24 hours by a submersion victim.[7]

Children and young adults predominate in drowning morbidity and mortality statistics,[8,9] with toddlers and teenage males the most frequent age and sex groups. Factors common to drowning incidents appear in Table 1. Risk-taking behavior and use of alcohol are common findings in teenage male victims of drowning and near-drowning.[1-3,10] Freshwater drownings outnumber saltwater drownings in most series.[11,12] My personal experience in more than 1,500 submersion incidents suggests that the vast majority of such accidents could be prevented by improved education and supervision of swimmers.

Table 1. Factors Common to Drowning Incidents*

Age:	Toddlers, teenage boys
Location:	Home swimming pools
Gender:	Males predominate in every age group
Race:	Black children most at risk
Drugs:	Alcohol in particular
Trauma:	Secondary to diving, falls, horseplay, and, recently, power-boating

*From information in Reference 7.

Dr Nemiroff is Captain, U.S. Public Health Service, and Senior Medical Officer, U.S. Coast Guard Training Center, Petaluma, California.

A version of this paper was presented by Dr Nemiroff on October 3, 1991, during the RESPIRATORY CARE Journal Conference on Emergency Respiratory Care, held in Cancun, Mexico. The poem "River Dragon" is reprinted, with permission, from "Updwellings," a newsletter from Michigan Sea Grant, a program of The University of Michigan, Ann Arbor, Michigan.

Reprints: CPT Martin J Nemiroff MD, U.S. Coast Guard Training Center, Health Services Division, Petaluma CA 94952-5000.

Pathophysiology

The sequence of events in near-drowning in animal studies[13] and from my own retrospective interviews of survivors is summarized in Table 2.[14] Variations in this sequence have been noted in special situations. Gooden noted that toddlers who accidentally enter cold water become suddenly immobilized and float facedown with arms and legs outstretched on the surface, with little apparent struggle.[15] In profoundly cold water, the involuntary gasp is enhanced and the conscious breath-hold time is markedly reduced.[16,17] If hypoventilation occurred on the water's surface, hypoxia may cause sudden loss of consciousness before the hypercapnic drive to breathe is evident. This has been called shallow-water blackout.[18] It has been estimated that 10% of near-drowning victims experience severe laryngospasm with the first in-rush of cold water, and fluid does not enter the trachea.[19] This has been called dry drowning.[6,7] A more common occurrence is that water enters the larynx, leading to a reflex parasympathetic-mediated constriction of the airways.[6]

Table 2. Human Near-Drowning Sequence*

1. Violent struggle to reach the surface
2. Period of calmness and apnea
3. Swallowing of large amounts of fluid, followed by vomiting
4. Gasping respirations and aspiration
5. Convulsions, coma, and death-like appearance

*From information in Reference 14.

Hypoxemia occurs in virtually every submersion accident, resulting from decreased ventilation. Aspiration of fluid is followed by apnea, progressive hypoxemia, profound acidosis,[20] arrhythmias, and death. One series of arterial blood gases from 51 hypothermic nearly drowned persons is summarized in Table 3. The duration of submersion that a human being can withstand without permanent brain damage is unknown. I witnessed and treated a 3-year-old girl who was submerged for 60 minutes in water of 3.3°C (38°F) temperature and made a full neurologic recovery. Reports of other long-duration (> 7 minutes) submersions with survival are mostly limited to cold-water situations (> 21°C, 70°F).[5,21,22] The pathophysiology in cold-water incidents differs from that in warm-water situations. The majority of drownings and near-drownings in the United States occur in backyard heated swimming pools, and therefore neurologic survival time is usually limited to a 4- to 6-minute airless period, and morbidity is high.[11] Controversy exists as to whether the mammalian-diving response plays a role in the neurologic recovery of the long-submersion cold-water victim. A dichotomy exists between the physiologists and some clinicians, with the physiologists believing that there is little proof of its role in survival,[23-25] and the clinicians believing that it is a good explanation for survival in newborn infants and toddlers.[4,14,15]

Table 3. Mean Arterial Blood Gases and pH in 51 Hypothermic, Nearly Drowned Individuals*

pH: 7.08
7.13 when corrected for body temperature

P_{aCO_2}: 59.64 torr
52.14 torr when corrected for body temperature

P_{aO_2}: 96.75 torr†
79.13 torr when corrected for body temperature

* From information in Reference 20.

† The majority of these patients were receiving 100% oxygen when the blood sample was drawn.

An additional controversy surrounds the question of the effects of seawater versus fresh water in the lung. Whether submersion occurs in seawater or fresh water, the primary pathophysiologic findings are profound and persistent hypoxemia and metabolic acidosis.[20] Damage to lung surfactant occurs by denaturation with fresh water, or by washout with salt water—with resultant alveolar injury and pulmonary edema.[26] Animal studies show a return to normal P_{aCO_2} 1 hour after aspiration of fresh or salt water; however, hypoxemia persists.[27] Estimates of the amount of fluid aspiration in human beings are less than one might expect (10 mL of fluid per pound of body weight); therefore, lungs are not filled with aspirated water.[26,27] Consequently, the small volume of water aspirated does not affect the circulating blood volume.[26,28] Increases in capillary permeability lead to noncardiogenic (low-pressure) pulmonary edema and proteinaceous fluid loss in the alveolar spaces.

The gross appearance of lung in the drowned individual includes a frothy, white, slightly bloody foam, evidence of pulmonary edema; and petechial hemorrhages are seen in the lung and pleura.[27-29] Microscopically, changes are seen in the alveolar wall, with focal emphysema, and in the vascular endothelium, with swelling of the mitochondria.[30] If the submersion occurred in unclean water, ponds, sewage, swamp, or mud, a variety of pathogens may be seen. Our experience includes recovery of blue-green algae, *Aspergillus*, and *Pseudomonas* sp. from drowned and nearly drowned lung. The totality of these changes contributes to ventilation-perfusion mismatch and resultant hypoxemia. Differences between the effects of seawater and fresh water are therefore imperceptible to the clinician.

A major milestone in the understanding of near-drowning pathophysiology occurred with the findings of Modell, who reported in 1968 that the primary pathophysiologic disturbance that necessitated emergency treatment in human near-drownings was acute asphyxia with resultant hypoxemia and acidosis.[31] Modell reported that electrolyte changes in human beings were minimal and required little, if any, emergency treatment. Once again, little difference could be noted between freshwater and seawater cases.[31]

The First Responder

The recognition of a submersion victim is not difficult. Teaching the correct response is! A common appearance of the near-drowning victim is a cold, cyanotic, stiff person with poor circulation. He may be apneic, unresponsive, and have fixed, dilated pupils. On occasion, errors are made because of this deathlike appearance. Often, basic life support (BLS) is not begun, or, worse, the area is secured as a 'crime scene.' I have called this "death behavior." More correct behavior, termed "life behavior," begins with removal of the victim from the water. After a quick check of the pulse, and if the person was submerged less than an hour, BLS is instituted and help is called for. My experience, and that of others,[22] is that fixed, dilated pupils do not necessarily portend ultimate poor prognosis in hypothermic near-drowned individuals.[1,2] In contrast, Modell et al, in a 1976 paper,[12] reported no survivors who had fixed and dilated pupils; however, the victims were not significantly hypothermic. Water is an excellent conductor of body heat and cools the body 25 times faster than air of the same temperature.[32] Therefore, most of these victims are hypothermic, and they become more so as evaporation occurs and if wet clothes remain on the body after the victim is removed from the water. Their management includes ventilation, preservation of body heat by removing anything wet and replacing it with dry coverings, and gentle transport to a site of definitive medical care as soon as is practical.[33,34]

Controversy exists about airway-clearing maneuvers. Historically, airway clearing and resuscitation have taken many forms, including rolling the victim over a barrel or placing him on the back of a trotting horse. More modern recom-mendations have included "breaking" the patient (bending at the waist with the head down) or positioning. Current knowledge of the pathophysiology of near-drowning suggests that these methods are unlikely to drain the thick, proteinaceous pulmonary-edema fluid and affect the success of resuscitation. The Heimlich maneuver has been suggested by its inventor as a first-line treatment for every drowning victim.[35] Others feel that this manuever brings an increased risk of vomiting and aspiration, and that abdominal-thrust maneuvers should be reserved for airway obstruction only.[36,37]

In summary, the accepted standard of care that has evolved in the United States is to attempt resuscitation in any cold-water long-submersion near-drowning victim if he has been submerged an hour or less in water at 21°C (70°F) or colder.[34,38]

Cold-Water Near-Drowning and Hypothermia

Cold-water long-submersion victims have unusually high survivability, as shown in a series of consecutive cases studied by our group since 1975. Data were collected on each victim's age, locale and circumstances, underwater time, water and air temperatures, cardiopulmonary resuscitation efforts, water type and quality, and listing of any major injuries. The minimum emergency data required were approximate age of the victim, estimated underwater time, and water temperature. Table 4 shows the prognostic factors we have identified from this study.

Table 4. Prognostic Factors in Cold-Water Near-Drowning Survival*

Age: The younger the age, the better the survival (mean survivor age 11.2 y).

Underwater Time: The shorter, the better (range 7-60 min).

Water Temperature: The colder, the better (range 27-70°F).

Quality of CPR: Major breaks in technique or omissions reduced survivability.

Amount of Struggle: The less struggle, the better survival.

Water Quality: Better survival in clear water (translucent) than in swamp, mud, or other fluids (eg, liquid fertilizer).

Other Major Injuries: Other injuries markedly reduced survival regardless of injury (eg, blast, burn, head, or long-bone trauma).

Suicidal Intent: Victims seeking suicide by drowning did less well than matched victims of accidental submersion.

* Data from observations, by the author's group, of 100 cases of near-drowning during the period 1975-1990.

Treatment during Transport

The primary management of the near-drowning patient during transport is respiratory support. Immediate airway management is dictated by individual patient factors: level of consciousness, level of responsiveness, patency of airway, presence of secretions, vomiting, aspiration, or apnea. Airway management includes positioning and oro-, naso-, or endotracheal intubation, preferably with supplemental oxygen at an F_{IO_2} of 1.00. Assessment of circulation dictates whether chest compression is necessary.

Rewarming attempts are unnecessary during most transports; in fact, if made, they may increase cardiac irritability. Remaining body heat is conserved by removing wet garments and covering the high heat-loss areas of the head and neck, axillae, and inguinal area with warm, dry coverings if possible[39]—much as one would use to transport a neonate.

Vital signs, including rectal temperature, should be monitored closely. It should be noted that body temperature often falls during transport of these patients. This is a precarious situation, with ventricular irritability highly likely to occur. Should a cold-induced ventricular arrhythmia occur, it is very difficult to normalize with countershock. Advanced cardiac life support (ACLS) procedures and protocols for hypothermic individuals limit countershock to one shock in the field, and they do not advocate using more than lidocaine and bretylium until definitive rewarming can be accomplished.[40,41] Spontaneously breathing patients should be monitored with pulse oximetry during transport.

Treatment at the Hospital

The initial in-hospital management of the near-drowned victim is an extension of the prehospital care. Hypoxemia persists after aspiration of fluids due to the alveolar capillary damage, with continued intrapulmonary shunting.[28] Of pertinence here is the respiratory evaluation and management algorithm recently published by Shaw,[27] which appears here as Figure 1.

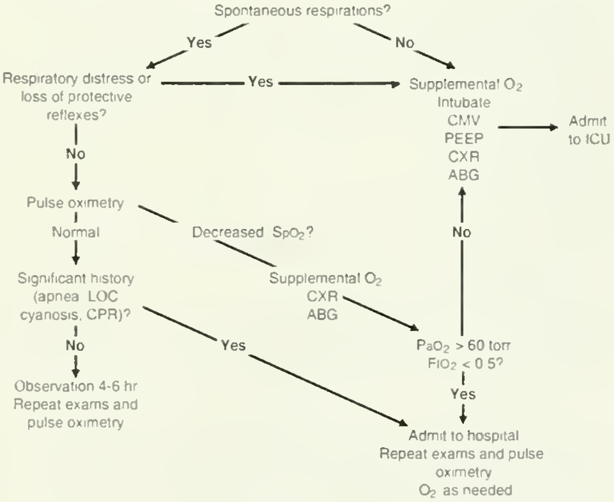
Spontaneous respirations?
Yes
No
Respiratory distress or loss of protective reflexes?
Yes
Supplemental O2 Intubate CMV PEEP CXR ABG
Admit to ICU
No
Pulse oximetry
Normal
Decreased SpO2?
No
Significant history (apnea LOC cyanosis, CPR)?
Supplemental O2 CXR ABG
No
Yes
PaO2 > 60 torr FIO2 < 0.5?
Observation 4-6 hr Repeat exams and pulse oximetry
Yes
Admit to hospital Repeat exams and pulse oximetry O2 as needed

Fig. 1. Respiratory evaluation and management of the near-drowning patient (with stable cardiovascular condition). ABG = arterial blood gas determination, CXR = chest radiograph, ICU = intensive care unit, LOC = loss of consciousness, S_{pO_2} = oxygen saturation via pulse oximetry, CMV = continuous mechanical ventilation, and PEEP = positive end-expiratory pressure. (Reprinted, with permission, from Reference 27.)

The level of hypoxemia approximates the alveolar damage from aspiration. Chest-radiograph evaluation can be a determinant of the eventual magnitude of therapy; however, normal chest radiographs do not rule out the possibility of deterioration in the first 24 hours.[42] In general, the nadir of radiographic change occurs within 24 hours in the typical case, with gradual resolution over the ensuing 7 days if superimposed pneumonitis is not a complication.[19,43] Early intubation and the use of positive end-expiratory pressure (PEEP) should be considered for patients unable to maintain a $P_{aO_2} >$ 60 torr with an F_{IO_2} of 0.50 or lower.[28] Traumatic pulmonary injury is seen frequently in near-drowning victims, and barotrauma may result from mechanical ventilation with PEEP. The complications and problems faced in the management of near-drowning are summarized in Table 5.[43,44]

Table 5. Complications of Near-Drowning in the Acute and Subacute Hospital Period

Hypoxemia
Acidosis
Hypothermia
Hypovolemia
Cerebral anoxia
Hemolysis
Diffuse intravascular coagulation (DIC)
Renal failure
Hepatic failure
Necrotic bowel
Sepsis
Pneumonias with unusual etiologies: fungal, algal, and bacterial
Febrile illness: *Francisella philomiragia*[43]
Brain abscess: *Pseudallescheria boydii* (up to 6 months later)[44]

Rewarming should proceed concomitantly with all other treatment modalities. Due to the loss of body heat by convection, conduction, radiation, and evaporation, these victims are often hypothermic.[5,22] Efforts should be made to protect the high-heat-loss areas, including the head, neck, axillae, and inguinal region.[39] Rewarming techniques include airway rewarming; administration of heated intravenous solutions; heated lavage of the gastric, intrathoracic, pericardial, and peritoneal spaces, as well as the urinary bladder and rectum.[45] External heat has been applied by heating blankets, warm baths, and near-total immersion in a Hubbard tank. Extracorporeal circulation with total cardiopulmonary bypass has been successful.[22] The method of choice is often dictated by the level of core temperature and the availability of the aforementioned techniques.

Summary

Submersion incidents are common, and they carry a high morbidity and mortality. Near-drowning often strikes healthy young children and teenagers prone to risk-taking. Recent advances in our understanding of the pathophysiology of drowning have led to improved respiratory management, leaving other less treatable complications to take their toll—cerebral death and diffuse intravascular coagulation (DIC), for example. The most evident recent advances in prehospital care, such as aggressive dive rescue, development of emergency medical technicians, and public cardiopulmonary resuscitation training have made a dent in the fatality statistics. Further effort should be directed toward training of emergency department physicians and routine use of available protocols for management of hypothermia and near-drowning.

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Nemiroff Discussion

Porter: At what point do you decide to go beyond inhalation warming or is that all that is ever required? Have you gone to cardiopulmonary bypass?

Nemiroff: We move to cardiopulmonary bypass very quickly if we have it available. Many of these patients were in remote areas of Alaska—with cardiopulmonary bypass 250 miles away. Therefore, it was some hour and a half to cardiopulmonary bypass. Even in that setting, we have done it successfully more than once; so, yes, we have. What is our criterion for cardiopulmonary bypass? Availability.

And our perfusionists are delighted to see these cases. This is a wonderful skill that they have already developed to a high degree (no pun intended). They rewarm these patients and measure just about every ion. We know where we stand very quickly; so we highly encourage cardiopulmonary bypass. Now, that may be using a howitzer where a .22 might be effective. If we're having some response to rewarming (for example, a bradycardic rhythm), I don't consider going to bypass. But if you have a patient with an inert heart, a very cold patient who is not responding to your favorite rewarming technique—of course, we could discuss rewarming techniques for 2 weeks—if you have chosen your favorite technique and it isn't working, then I would move toward bypass very quickly. I would alert the perfusion team to that fact as you are resuscitating the patient, so they can be thinking in terms of fitting the rewarming patient into their regular standard schedule.

Thompson: Would you describe your rewarming technique?

Nemiroff: Yes, I use 105-108°F, 100% humidified oxygen, and I've done it different ways—a $10 homemade way with a thermos bottle and a coil (running the oxygen through a heated thermos bottle). I've also modified a mainline nebulizer on a mechanical ventilator. I think I used an Ohio, drove it beyond its standard range, and had the company modify it to heat to 105-108°. I measured the temperature at the mouth. With every one of these, we measure at the endotracheal tube when we're using heated oxygen. At least 3 other companies have agreed to increase the highest temperature limit on their equipment so we could achieve 105-108°. I don't want to go above 108°, or much below 105°.

Weaver: When do you not resuscitate? Or, do you always? I mean, what criterion do you use for deciding not to attempt resuscitation?

Nemiroff: The criterion we use for 'Do not resuscitate' is submersion longer than 60 minutes. Now, if you're an emergency medical technician (EMT), you are very rigid, and you say, "60 minutes, it's over." I may go to 65, but we have had no meaningful survivals beyond that time. I cannot find a series with full survival of patients submerged beyond 60 minutes. So, pretty clearly, 60 minutes is the current standard. We do not resuscitate if we feel that we have major loss of limb, open-brain injuries, or other conditions that you would not resuscitate under ACLS and other standard protocols.

Wilson: You mentioned, I believe, that the colder the water the better the survival. Is there indeed a relationship between initial core temperature and lower core temperature in survival?

Nemiroff: Yes, there is. There have been no meaningful initial core temperatures much below 60°F. A good marker, by the way, is the serum potassium level. There have been no survivals in hypothermic patients with markedly elevated serum potassiums. It's one of the new ways of getting a quick prognostic indication in a severely hypothermic patient. So, if the elevated serum potassium is from the hypothermia alone, it is a good marker, which was recently reported in *JAMA*.[1]

[1] Schaller MD, Fischer AP, Perret CH. Hyperkalemia: a prognostic factor during acute severe hypothermia. JAMA 1990;264(14):1842-1845.

Wilson: As you're in the process of resuscitating an individual, is there a critical core temperature at which you can comfortably discontinue CPR—once you reach it and still have no response?

Nemiroff: 92°F. If we reach into the 90s, preferably 91° or 92° core temperature, and we're still not getting a cardiac response, then that's a decision point. We stop the resuscitation. That takes about 3 hours. These are lengthy resuscitations (3-3.5 hours) that bring a standard emergency department to its knees in terms of backup of patients and everything else. When we get to 91-92° without response, then we generally cease.

Wilson: One final question—105°, in terms of airway temperature, is, I think by many standards, fairly conservative. Is there some reason that you limit there? I mean, in operating rooms we'll use temperatures higher than that (at least measured at the airway) for maintenance of core temperature, which, as you know, isn't terribly effective.

Nemiroff: This was studied by Steinman, Chaney, and Collis in the 1970s.[1] They reviewed animal work that showed that bronchial and tracheal mucosa are injured by temperatures higher than 108°. So all our studies have been limited to < 108°. I don't use temperatures higher than that because our resuscitative literature was based on those initial studies.

[1] Steinman AM, Collis ML, Chaney RD. An experimental study of practical rewarming methods. National Assn for Search and Rescue. PO Box 8100, Salt Lake City, Utah. Sept 9-12, 1976.

Reines: Because you're mentioning a national standard, I'm a little concerned. What's the denominator? You have 51 survivors in the numerator. What's the denominator? What's the cost? What's the risk of ending up with a vegetable if you resuscitate? You said 1 hour—and was it 70° immersion temperature, or . . .?

Nemiroff: I defined that series as cold-water, long-submersion victims and tried to make it very clear that these are not backyard pool Southern California-type cases, which account for 7,000 out of 8,000 of our drowning patients per year. I'm talking about, in this country, a subseries of approximately 1,000 per year of cold-water cases. Of that 1,000, there is a subset of long-submersion cases.

Reines: With 51 survivors over how many years?

Nemiroff: I've been studying this since 1975. These are my personal cases (unpublished data). I haven't seen all 1,000 cases in the United States.

Reines: I'm just trying to see if we're talking about 1%, 10% . . ..

Nemiroff: I can only give you my personal experience because there are no national statistics. From my personal statistics of 51 long-term survivors, I have 3 patients who are chronically hospitalized. Victims either die or survive, and 3 out of 51 is a small number. I can give you my personal series, but I cannot relate this to anything nationally because there are no statistics—and I've searched and wanted and waited for some. Exactly how many people drown in this country (*fait accompli* deaths) is not known. There is only an estimated statistic.

Hess: My question relates to blood gases in the management of hypothermic patients. Do you manage acid base and oxygenation based upon the temperature-corrected values or the uncorrected values? That's an area of some controversy, and I know we banter about that in our own emergency department.

Nemiroff: I list both of them in my illustrations.

Hess: Well, that's the way we do it, too. That's how we get around it.

Nemiroff: That's how I skirt the controversy, by showing both. What I really do is use the measured blood gas values, not the temperature-corrected values. But you caught me hedging, by my slide.

Hess: There's good support for that.

Haponik: Would you please comment about the rules or lack of rules for corticosteroids, intracranial pressure monitoring, and barbiturates, in this setting. There seems to be a mythology about some of them.

Nemiroff: The current review of the literature suggests that corticosteroids are of no benefit.[1-3] I've used them in a significant portion of my series and then stopped using them when the literature made it apparent that we may actually be causing some damage and no benefit. Initially I felt that induced barbiturate coma and induced hypothermic coma had tremendous value in my hypothermic patients, but the larger series that are now being presented show that there is no real statistical improvement in survival using barbiturate-induced and hypothermia-induced coma. Looking at those series critically, which I did, I could not tell which were hypothermic cold-water drownings and which were warm-water drownings because it wasn't listed in the papers. The source of the patients was not clear, and I had to assume on the basis of where the paper had been written what the basic population was. So, I don't have an answer for you except to say that the literature is now saying not to induce barbiturate or hypothermic coma. I have a gut reaction that it may still be effective in the cold-water cases, but I have nothing, really, to base my reaction on in terms of a controlled series. Given the opportunity, having a Richmond bolt, a neurosurgeon, and the skills available today, I would use it.

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Barnes: Has the Coast Guard settled on a standard for emergency ventilation? Are you intubating? Are you using bag-valve-mask, mouth-to-mask, mouth-to-mouth?

Nemiroff: We are no longer flying with flight surgeons. We fly with a basic EMT and a rescue swimmer. He may be one and the same person, and, therefore, we do not intubate. We bag-valve-mask almost entirely, unless there's a physician aboard, and that's a rarity in our current Coast Guard.

Barnes: In a way, I'm glad you said that because I want to ask you about gastric inflation and if that's a problem with bag-valve-mask breathing in these rescues.

Nemiroff: You mean in the GI tract? It's always a problem for us, and one way that we are very cautious is in educating our people not to lean on the abdomen when they're doing a resuscitation and to treat the airway very gently. As soon as we get the patient, we protect the patient with an endotracheal tube. In rare circumstances we fly with a paramedic who has ACLS background and training, and frequently he is prepared to intubate, and may do so. What would I like to see? I'll tell you privately.

Mathews: I was reading an article in preparation for this conference by Ashwal and associates from Loma Linda[1] that suggests that perhaps tracking levels of blood glucose and cerebral blood flow are predictive of one of the three outcome states from drowning (the outcome states being unimpaired survival, impaired survival, and death). They showed an inverse relationship between the level of blood glucose and the survival status. Do you have any comments on that?

1. Ashwal S, Schneider S, Tomasi L, Thompson J. Prognostic implications of hyperglycemia and reduced cerebral blood flow in childhood near drowning. Neurology 1990;40:820-823.

Nemiroff: Once again, the blood glucose in a hypothermic individual differs from that in a warm individual, and I don't know the series that you're mentioning, by letter and verse. I would have to know more about the patient population and have it separated for body temperature and the cause of the near-drowning. Then I would better be able to answer your question. As I said, I don't know the particular paper, but I could bet you that the series does not separate out subjects in terms of initial body temperature, the initial incident in terms of what the cause of the near-drowning was. It makes it very difficult to answer the question you're asking.

Mathews: OK. And, just as a follow-up, I came across an interesting article in *Annals of Emergency Medicine* in June of 1991[1] that cited an industrial drowning on an oil rig. I don't know if you're familiar with that particular article. A gentleman fell into a cleaning vat on an oil rig. It was water mixed with calcium salts. By the time they got him to the hospital, he was in pretty bad shape and actually had hypercalcemia secondary to ingestion. That brings up a question. Is it important for the rescuers, if possible, to bring a sample of the water into the hospital with them—a sample of the fluid, so we know what these people drowned in?

1. Fromm RE Jr. Hypercalcemia complicating an industrial near drowning. Ann Emerg Med 1991;201(6):669-671.

Nemiroff: I've requested that numerous times. For one reason—because I want to culture it in the laboratory at the same time I'm treating my patient. What I'm trying to do is to determine sensitivities in vitro at the same time that I'm treating a human being—to be able to change my therapy quickly in the first 24 hours if I see that I'm heading in the wrong direction. Yes, we do request a sample, and emergency personnel, wanting to be helpful in this setting, will run back and get me water temperatures and water samples for culture: they'll get me samples of almost anything that I need very quickly and bring them back to the hospital. Usually, the responding emergency agency will bring those samples to me quickly. Yes, I've found that useful.

There is a paradigm for that from Israel—reports of drowning in waters of very high salinity,[1] and those do lead to very major electrolyte changes. But that's such a rare circumstance.

1. Yagil Y. Near drowning in the Dead Sea: electrolyte imbalance and therapeutic implications. Arch Intern Med 1985;145:50.

Durbin: Do you reduce the immersion-time criterion for initiating resuscitation in the elderly? You mentioned that the elderly don't do as well. How would you change your management in this population?

Nemiroff: Well, I used to. I used to have what we call a game face, and my game face was pretty sad when I had an older victim. But we've had a 72-year-old, 30-minute submersion with full recovery, which in a sense makes us very uncomfortable because it makes what we teach probably incorrect. In other words, our predictive value is not always as good as we'd like it to be, and once again, we don't know enough, and we're learning every day.

Haponik: In their extensive Australian experience Pearn et al[1] have commented about the importance of the brief time to first gasp. In your extensive series, have you looked at that as a prognostic factor?

1. Pearn JH, De Buse P, Mohay H, Golden M. Sequential intellectual recovery after near-drowning. Med J Aust 1979;1(10):463-464.

Nemiroff: We have no data on that.

Smoke Inhalation Injury: Some Priorities for Respiratory Care Professionals

Edward F Haponik MD

Introduction

The varied respiratory complications caused by smoke inhalation epitomize the clinical challenges that confront respiratory care professionals. The presentations and management of upper-airway, tracheobronchial, and lung parenchymal injuries, their combinations, and potential superimposed iatrogenic problems require an extensive, practical knowledge of the pathophysiology of this injury and of basic therapeutic principles. The development of systemic collapse due to asphyxiants such as carbon monoxide or cyanide, the frequent presence of cutaneous burns, and the added vulnerability conferred by coexisting illnesses require meticulous attention to the details of daily care. Moreover, adaptations of management according to the rapidly changing needs of each patient, optimum use of current technologic support, and vigilant avoidance of iatrogenic complications are essential priorities.

Despite the frequency and gravity of smoke inhalation injuries, remarkably few reports address the demands that smoke inhalation injury imposes upon the respiratory care practitioners who have a central role in its management. These responsibilities may range from intubation and resuscitation of victims at the scene of a house fire, to the emergency room triage of victims of a hotel conflagration or industrial disaster, to assistance with diagnostic bronchoscopy, to performance of pulmonary function testing, to arterial blood gas analysis or CO-oximetry, to safe transportation of the patient for diagnostic testing or emergency surgery, to optimization of mechanical ventilator management of multiple changing physiologic derangements, and to avoidance of potentially fatal nosocomial pneumonia.

The degree of responsibility imposed upon respiratory therapists in meeting these diverse challenges merits particular comment. Although in some instances the respiratory therapist functions as a member of a large, well-integrated multidisciplinary team (eg, as in a tertiary care center's burn unit), in others the therapist must lead management efforts because he or she possesses the most familiarity and experience with the problem. The acuteness, pace, and unpredictability of potentially lethal complications and the tendency of fire-related disasters to occur during night or early morning hours (coinciding with periods of reduced staffing in medical centers) all serve to augment the pressures focused upon respiratory care professionals. Coordination of respiratory care activities with those of nursing staff and primary and consulting physicians is essential for optimum patient outcomes but sometimes difficult to achieve. Finally, appropriate documentation of monitoring and communication of details of each patient's respiratory status provide important, unique perspectives that enhance overall management.

Magnitude of the Problem

Smoke inhalation is a worldwide hazard with potentially lethal consequences.[1-16] Despite the successful use of smoke detectors and enhanced public awareness, each year approximately 2 million persons in the United States sustain burns requiring

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Dr Haponik is Professor of Medicine, Section on Pulmonary/Critical Care, Wake Forest University Medical Center, Bowman Gray School of Medicine, Winston-Salem, North Carolina.

A version of this paper was presented by Dr Haponik on October 3, 1991, during the RESPIRATORY CARE Journal Conference on Emergency Respiratory Care held in Cancun, Mexico.

Reprints: Edward F Haponik MD, Section on Pulmonary/Critical Care, Bowman Gray School of Medicine, Medical Center Blvd, Winston-Salem NC 27157-1054.

medical attention.[6] Approximately 4% of them ultimately require intensive care management, and from 6 to 10 thousand Americans die despite such supportive efforts. Not surprisingly, smoke inhalation injury is more often managed in urban centers (20-35% of fire victims) than in rural settings (10-15% of individuals), but such patients are encountered in virtually all respiratory care practices.[6]

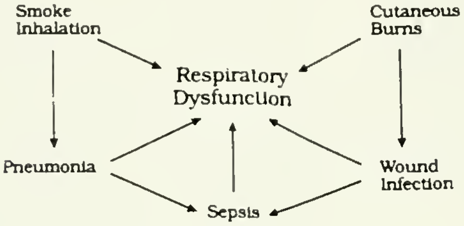

Fig. 1. Major interactions occur between smoke inhalation and cutaneous burn. Each of these problems can lead to profound respiratory dysfunction. Superimposed infections of the lung and skin can lead to further respiratory compromise. (Reprinted, with permission, from Reference 12.)

Although the mortality from smoke inhalation alone is low (0-11%), smoke inhalation in combination with cutaneous burns proves fatal in 30-90% of patients. With progress in the management of burn shock, renal failure, and wound sepsis, respiratory problems—inhalation injury and superimposed pneumonia—have emerged as the major causes of death. These interactions are outlined in Figures 1 and 2.[6,7,12] Recently it has been reported that the presence of inhalation injury increases mortality by 20% and that inhalation injury predisposes to pneumonia.[12-14] Pneumonia independently increases burn mortality by 40%, and the combination of inhalation injury and pneumonia leads to a 60% increase in deaths.[14] The impact of these complications is most apparent in individuals who otherwise who would have been expected to survive (when prognoses were estimated on the basis of the extent of the burns alone).[17] Children[18] and the elderly[19] are especially vulnerable to these problems because of limited physiologic reserve.

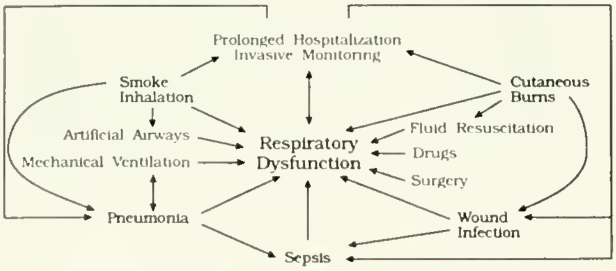

Fig. 2. Several nosocomial problems are typically superimposed upon the potentially lethal interaction between smoke inhalation injury and burns. Therapeutic modalities for either problem may have adverse effects on respiratory dysfunction. Prolonged hospitalization associated with severe injury predisposes to still further respiratory problems. (Reprinted, with permission, from Reference 12.)

In Thompson et al's major review,[18] patients with smoke inhalation injury had a 50% mortality compared with only a 4.1% fatality rate in burn victims without inhalation injury. Among individuals who require mechanical ventilatory support during burn management, nearly two thirds have sustained inhalation injury; the need for such support has been associated with a 92% mortality.[20] Prolonged hospital stay (with an approximate 2-week extension of hospitalization) and associated high costs of medical care (exceeding $300 million annually) add to the personal tragedies seen in this setting.[12]

Pathogenesis of Smoke Inhalation Injury

Smoke is a heterogeneous product and can evolve either through pyrolysis (smoldering, in a low-oxygen environment) or combustion (burning, with visible flame, in an adequate oxygen environment). An array of particulates and gases is generated, varying with the composition of the fuel and other factors, and represents the primary cause of respiratory injury.[21-26] Contrary to popular belief, direct lower respiratory tract damage is seldom caused by heat alone, and the terms "respiratory burns" and "burned lung" are generally misnomers. Catastrophic upper-airway injury can sometimes occur solely due to thermal effects, but, in most circumstances, the airways are remarkably efficient as a "heat sink."[27] An important clinical exception is associated with steam inhalation. Because of its increased heat-carrying capacity, steam can cause true thermal pulmonary damage. Recently, true lung burns have also been found in association with inhalation of the crack form of cocaine and ignition of the ether vehicle.[28]

Some of the more commonly encountered products of incomplete combustion are outlined in Table 1.[23,29,30] Carbon monoxide (CO) and acrolein are particularly noteworthy in residential fires, but there has been increasing appreciation that cyanide generated from nitrogen-containing polymers has an important role in early incapacitation and/or mortality.[31-33] Cyanide, a cytochrome poison, has been implicated in the deaths of up to one fourth of smoke inhalation victims in some series. Most early fire deaths are believed to be due to carbon monoxide intoxication (approximately 60-80%) rather than to cutaneous burns (approximately 11%).[34] Recognizing tissue hypoxia caused by this gas (despite a normal or high arterial oxygen partial pressure, P_{aO_2}) requires a high index of suspicion. CO, with its affinity for hemoglobin 250 times that of oxygen, leftward shift of the oxyhemoglobin dissociation curve, and potential effects upon cytochromes, markedly compromises oxygen transport and utilization.

Table 1. Common Toxic Gases Generated during Fires*

Source	Product
Polyvinylchloride (PVC)	Hydrogen chloride,† chlorine, phosgene, carbon monoxide
Wool, silk, nylon, polyurethane, melamine resins	Hydrogen cyanide,† ammonias, isocyanates
Nitrocellulose film, fabrics	Oxides of nitrogen
Wood, cotton, paper petroleum products	Acrolein,† acetaldehyde, acetic acid, carbon monoxide†
Fluorinated resins	Hydrogen fluoride, hydrogen bromide
Sulfur-containing compounds	Sulfur dioxide
Petroleum products	Benzene

*Adapted, with permission, from Reference 6.
†Commonly implicated.

Products of polyvinylchloride (PVC) pyrolysis also are important offenders because of the ubiquity of such polymers in modern construction and furnishings.[35] Intense, but unrecognized, inhalation exposures to phosgene, chlorine, and hydrochloric acid may occur during not only the fire itself, but also during the 'overhaul' or cleanup phase after the fire has apparently been extinguished. Health professionals involved in the field management of smoke inhalation victims must also recognize the potential for serious personal injury during this seemingly safe period and take appropriate precautionary measures.

Although in some instances the particular offending agent may be obvious, in most fires precise identification of the nature of the inhaled toxin is not feasible. The heterogeneity of fire scenarios also hinders efforts to develop models of inhalation injury, but observations in a number of experimental preparations demonstrate events that are markedly similar to clinical observations in humans.[36-43] Interestingly, these findings also parallel pathogenesis of acute lung injury in other settings. Inhalation of airway irritants results in direct mucosal damage. Depending upon the concentration of the agent, its water solubility, exposure and duration, anatomic levels affected and severity of airway injury sustained may vary.[29,30] Inhalation of gases with high water solubility (eg, ammonia, sulfur dioxide, hydrogen fluoride) predisposes to proximal, upper airway injury. By contrast, agents with lower water solubility (eg, hydrogen chloride, chlorine, phosgene, oxides of nitrogen) may be more insidious in their clinical presentation. Deeper penetration of the lung may be more likely to produce alveolocapillary membrane damage that may have a delayed clinical onset. Most inhaled irritant gases result in an oxidant-mediated epithelial injury, with associated denudation of the epithelial surface, increased permeability, and acute inflammation.[12] The roles of lipid peroxidation and, in particular, of granulocyte products including proteases and toxic oxygen free radicals are being investigated vigorously.[44-47] Modulation and augmentation of these cellular events by humoral mediators (including complement, serotonin and histamine, tumor necrosis factor, interleukins, and products of arachidonate metabolism) appear to be important.[48-54] The heterogeneity of smoke inhalation injury, its acuteness, and the essential focus of management on support of the patient have hindered delineation of the exact roles of these factors in humans. Progress in this area is essential for development of worthwhile pharmacologic interventions in the future.

Major physiologic effects of acute smoke inhalation are summarized in Table 2.[6,12] These relatively nonspecific events have direct implications for the clinical problems that must be addressed at the bedside. Reduced thoracic compliance accompanies interstitial and alveolar edema and/or extensive chest-wall burn, increases work of breathing, presents major problems in mechanical ventilation, and markedly increases the likelihood of barotrauma. Acute airway inflammation and edema are accentuated by increased bronchial blood flow. Bronchorrhea and anatomic airway obstruction by inspissated casts of carbonaceous material markedly increase requirements for suctioning and predispose to major atelectasis. Airway resistance increases not only from such mechanical obstruction but also from bronchoconstriction; the clinical presentation may resemble an acute asthma attack. Physiologic dead space is increased as a consequence of inhomogeneous ventilation. Because acute smoke inhalation also results in the immediate inactivation of surfactant,[55] acute microatelectasis occurs with resulting ventilation-perfusion mismatch.[56] In severe injuries, physiologic shunt leading to profound hypoxemia and acute microvascular injury with increased transvascular fluid flux produce a clinical picture of the adult respiratory distress syndrome (ARDS). Experienced respiratory therapists will recognize that this sequence of events is similar to manifestations of acute lung injury in other settings and probably represents the lung's only response to a catastrophic insult. The severity of each of these problems (together with their interactions with the effects of coexisting burns, fluid resuscitation, and other acute or chronic illnesses) markedly increases the challenges and hazards of management.

Table 2. Physiologic Effects of Acute Smoke Inhalation*

Decreased thoracic compliance†
Increased bronchial blood flow
Increased airway resistance†
Increased dead space†
Surfactant inactivation†
Hypoxemia†
Increased work of breathing†
Pulmonary hypertension†
Increased lung lymph
Increased lymph protein

*Adapted, with permission, from References 6 & 12.
†Experimental models and burn victims.

Major Acute Respiratory Complications

The principal respiratory sequelae of smoke inhalation and burns are summarized in Figure 3 and Table 3.[6,7,12] Recognition of separate, but often

Table 3. Major Respiratory Complications of Burned Patients*

- Inhalation injury†
  - Asphyxia
    - Hypoxia
    - Carbon monoxide, cyanide intoxication
  - Direct respiratory injury
    - Airway responses
      - Upper-airway obstruction
      - Tracheobronchitis
      - Bronchiolitis
      - Atelectasis
    - Alveolocapillary membrane injury (high permeability pulmonary edema)

- Effects of cutaneous burns
  - Pulmonary dysfunction
  - Chest-wall restriction (eg, circumferential thoracic burns, soft tissue edema)
  - Upper-airway compression (eg, circumferential cervical burns)

- Combined smoke inhalation and cutaneous burns

- Concomitant chest trauma

- Nosocomial/iatrogenic problems
  - Hydrostatic pulmonary edema (over-resuscitation)
  - Pneumonia (aerogenous, hematogenous)
  - Sepsis-related permeability edema
  - Drug induced
    - Hypoventilation (eg, morphine)
    - Hyperventilation (eg, topical mafenide)
  - Pneumothorax
    - Ventilator-induced barotrauma
    - Central venous-access procedures
  - Pulmonary embolism
  - Complications of artificial airways
  - Aspiration

- Exacerbation of pre-existing cardiopulmonary disease
  - Cardiogenic pulmonary edema
  - Chronic obstructive pulmonary disease

* Adapted from References 6, 7, and 12.
† Predominately due to smoke, but, in selected circumstances, due to direct thermal injury (eg, steam inhalation, free-basing explosions).

coexisting, effects of asphyxia (primarily due to CO and cyanide intoxication), airway damage (both upper airway and tracheobronchial), and injury to the alveolocapillary membrane (pulmonary edema) provides a framework for management priorities. The relative frequencies of these problems and their interactions are shown in Figure 3.[6,7,12] As can be seen, the likelihood of combined respiratory problems increases profoundly in the presence of cutaneous burns, accounting for a lethal impact that appears to increase in a synergistic way.

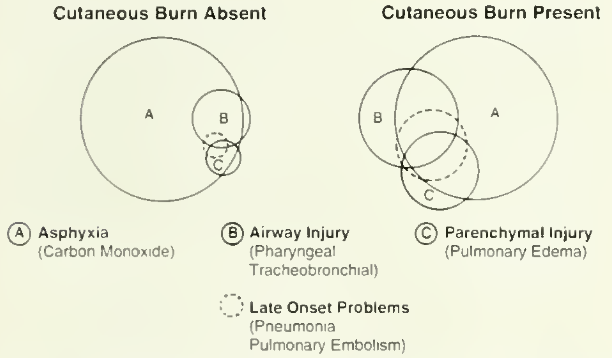
Cutaneous Burn Absent
Cutaneous Burn Present
A
B
C
Ⓐ Asphyxia (Carbon Monoxide)
Ⓑ Airway Injury (Pharyngeal Tracheobronchial)
Ⓒ Parenchymal Injury (Pulmonary Edema)
Late Onset Problems (Pneumonia Pulmonary Embolism)

Fig. 3. A spectrum of upper-airway, lower airway, and lung-parenchyma respiratory complications may accompany smoke inhalation. Their frequency and interactions are accentuated in the presence of surface burns. (Reprinted, with permission, from Reference 6.)

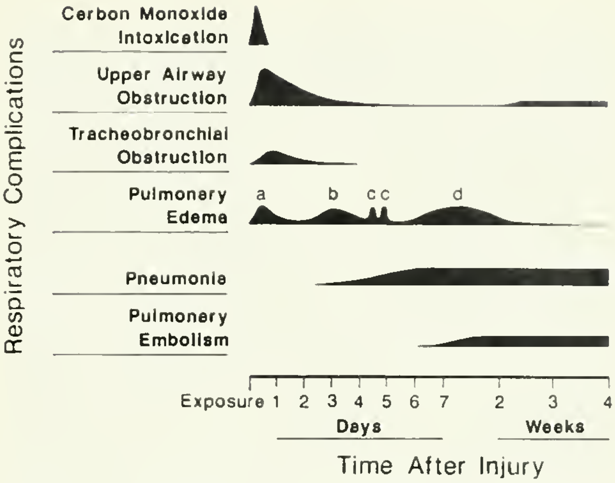

Fig. 4. A general sequence of respiratory complications has been identified following smoke inhalation and can assist in establishing diagnostic and therapeutic priorities. See text for details. (Reprinted, with permission, from Reference 7.)

Clinical experience has suggested that the relative likelihood of specific respiratory complications can be related to the time that has elapsed since exposure to smoke (Fig. 4).[6,7,12] The arbitrary classification of problems as occurring early (during the first 24 hours after inhalation), intermediate or delayed (Days 1-5), and late (beyond 5 days after exposure) is practical and useful. However, it must be recognized that global problems may coexist at the initial presentation of severe inhalation injury. Moreover, supervening respiratory compromise on the basis of other problems (eg, congestive heart failure with cardiogenic edema due to myocardial ischemia, overhydration due to fluid resuscitation, exacerbation of underlying obstructive lung disease, or aspiration pneumonia) can alter this course. CO and cyanide intoxication, acute upper airway obstruction due to pharyngeal edema, and tracheobronchial obstruction are typically early complications. Alveolar edema may occur at any time after exposure, and its causes vary considerably. As in patients with other acute lung injuries, nosocomial pneumonia and pulmonary embolism are major late-onset problems; predispositions are similar to those seen in other patients with respiratory failure, but are accentuated by the presence of cutaneous burns. Finally, major atelectasis, exacerbation of pre-existing cardiopulmonary disease, or pneumothorax may occur abruptly at any time.

Because the clinical manifestations of these problems (eg, dyspnea, altered gas exchange, reduced thoracic compliance) often may overlap, appreciation of the potential causes of a patient's acute deterioration and prompt diagnosis and therapy require familiarity with this natural history. Interpretation of such presentations may be complicated further by effects of treatment; for example, narcotic analgesics may markedly depress ventilation, whereas topical antimicrobial therapy with mafenide (Sulfamylon), a carbonic anhydrase inhibitor, or the late onset of sepsis may increase minute ventilation in the absence of primary lung injury.[57] Frequently, the recorded bedside observations of the respiratory therapist are key in differentiating among possible etiologies as a patient's apparently acute decompensation is investigated.

Acute upper airway obstruction (UAO) occurs in approximately one fifth to one third of hospitalized burn victims with inhalation injury and is a major hazard because of the possibility of rapid progression of mild pharyngeal edema to complete upper airway occlusion with asphyxia.[58,59] While heat and the effects of particulate and gaseous combustion products are the primary factors, additional predispositions have included the aspiration of hot liquids (especially in children),[60] flash exposures (such as can occur during the priming of carburetors with gasoline),[61] and ignition of petrolatum hair products.[62] The worsening of upper airway edema is most marked in supraglottic structures. Serial nasopharyngoscopic observations demonstrate obliteration of the aryepiglottic folds, arytenoid eminences, and interarytenoid areas by boggy edematous tissue that prolapses to occlude the airway.[63,64] If surface burns require rapid administration of resuscitative fluid, these changes can be accentuated. Burns of the neck may, especially in children, cause unyielding eschars that externally compress and obstruct the airway. As in other settings, trauma from endotracheal tubes (ETTs) presents yet an added predisposition to upper airway obstruction in this setting. Remarkably, the extent of danger presented by these problems is too often underestimated by the inexperienced. Some patients may have obvious signs and symptoms heralding upper airway compromise, but in others the course can be remarkedly insidious. Tragically, death still occurs from UAO despite the fact that it has been noted to be the most treatable of respiratory complications of smoke inhalation.[8,65,66] Thus, the adage "When in doubt, intubate" remains a well-supported guideline in this clinical setting.

Pulmonary edema may be multifactorial in its origin (Fig. 4). Noncardiogenic, high-permeability edema (ARDS) may result from alveolocapillary membrane injury by smoke, but contrary to popular belief is relatively uncommon after smoke inhalation or cutaneous burns alone. However, severe smoke exposures, burn shock, infusion of blood products, or, in particular, the late onset of hospital-acquired sepsis all may lead to ARDS. Cardiogenic edema and overhydration during fluid resuscitation or surgical procedures are other well-recognized hazards. Severely reduced oncotic pressure associated with protein losses from large surface burns also predisposes to alveolar flooding, and it has been recognized that mechanical factors may also contribute to pulmonary edema. The latter problem has been associated with acute upper airway obstruction, typically identified immediately after its relief by intubation or tracheostomy.[67,68]

The timing of pulmonary edema and carefully sought radiologic clues may be especially helpful in differential diagnosis.[69] Estimates of lung water have been of experimental interest but have not yet had a major practical role in resolving this dilemma.[70,71] Permeability edema with ARDS due to intense toxic gas inhalation, cardiogenic edema, and overhydration edema may all occur as early problems (a in Fig. 4). Hypervolemia during the period of remobilization of burn edema fluid and overhydration typically have a later onset (b and c in Fig. 4). Late-onset ARDS in patients with cutaneous burns is most often caused by sepsis (d in Fig. 4). Hemodynamic monitoring may be essential to clarifying these problems but at the potential cost of infectious complications.

Diagnosing Acute Inhalation Injury

The diagnosis of inhalation injury is generally based on the recognition of the spectrum and time course of 'classic' clinical predictors and respiratory problems, and the selective use of diagnostic tests (Table 4).[12,72-76] This complementary information facilitates early diagnosis and design of a planned, elective approach to therapy rather than an 'after the crash' approach.

Clinical Predictors

The history of exposure in a closed space, often with unconsciousness or entrapment of the victim, suggests that a more intense inhalation exposure has occurred. Altered sensorium can be associated with acute alcohol or drug intoxication but should strongly suggest severe hypoxemia, CO and/or cyanide intoxication, or coexisting head trauma.[77] The presence of any respiratory symptoms and signs must not be disregarded. Hoarseness, stridor, altered voice, painful swallowing, and obvious pharyngeal edema on physical examination are especially important because they connote acute upper airway obstruction. Cough, chest pain, or hemoptysis all may signal serious illness. Signs of inhala-

Table 4. Major Considerations in Clinical Diagnosis of Smoke Inhalation Injury*

- Spectrum and time course of respiratory problems
- Classic clinical predictors
  - Exposure characteristics
    - Closed space setting, entrapment
    - Unconsciousness
    - Known inhaled toxin
  - Respiratory signs and symptoms
  - Facial, cervical burns
  - Carbonaceous sputum
- Objective diagnostic tests
  - Measurement of asphyxiants (carboxyhemoglobin, cyanide)
  - Arterial blood gas analysis
  - Chest radiography
  - Direct airway observation (laryngoscopy or nasopharyngoscopy, bronchoscopy)
  - Radionuclide scanning
  - Pulmonary function testing (volume-time curves, complete flow-volume curves)

* Adapted from References 6, 7, and 12.

tion injury are less often present at the time of presentation: but cyanosis, tachypnea, stridor, wheezing, and crackles should be regarded as indicators of established injury. The relative frequencies of these signs and symptoms vary considerably among published reports, largely related to the nature of the reporting center and the time at which patients are initially evaluated.

The presence of facial burns is a 'classic' predictor of inhalation injury and requires special note.[78-82] This sign has only a variable relationship to respiratory injury because of the very high frequency of mild facial burns. Among all 'comers.' neither the positive nor negative predictive values of facial burns are sufficiently high to diagnose or exclude serious respiratory damage.[79] However, involvement of the so-called respiratory area of the nose and lips identifies an especially hazardous situation that must be recognized. Such a distribution of burns marks an inability to escape flame and is typically associated with extensive burns and the rapid progression of upper airway edema. This degree of facial and cervical involvement leads to eversion of the lips and immobilization of the jaw. These developments markedly limit (and even prevent) opening of the mouth, and may preclude even blind intubation. The patient's inability to clear pooled saliva or bedside observation of worsening hoarseness, sore throat, or painful swallowing should suggest progressing pharyngeal edema. In some individuals without obvious stridor, this sign is elicited readily by having the patient perform simple panting maneuvers. As noted previously, with large burns (and large requirements for fluid resuscitation), the rapid evolution of upper airway obstruction makes early elective endotracheal intubation not only prudent but mandatory. The experienced respiratory therapist must often instruct physicians in-training about the extent of danger and need for immediate response.

Carbonaceous secretions represent another classic sign of smoke inhalation injury that is a less exact predictor of the presence or severity of injury than is popularly believed.[12] Such viscid material may be an important cause of central airway obstruction or atelectasis, and demands mechanical airway clearance. Otherwise, these secretions should be regarded as a marker of exposure to smoke; they should heighten clinical suspicions, but they do not establish either the diagnosis of lung injury or its prognosis.

Laboratory Studies

During acute evaluations performed at the scene of exposure and at some community hospitals, initial diagnosis may be based exclusively upon historical clues and physical examination. However, widely available diagnostic tests offer valuable confirmatory information. Respiratory therapists are generally active in assisting with, performing, and/or interpreting these assessments. Moreover, when transportation of the patient to tertiary centers or to other locations in the hospital is necessary, the therapist is typically charged with accompanying unstable individuals and assuring their respiratory support.

Carbon monoxide intoxication is a major early threat to smoke inhalation victims. Although a number of available tables relate clinical signs and symptoms to particular arterial carboxyhemoglobin (COHb) levels, the variability among patients and the importance of this information requires that COHb measurements be made in every smoke inhalation victim.[12] These results are influenced by the time elapsed since exposure and the empiric oxygen therapy that one hopes has already been administered to inhalation victims in the field and during transport to the medical facility. Identifying elevated COHb levels is not only key to evaluation and therapy of carbon monoxide intoxication, but also may implicate important exposures to other toxic products (eg, cyanide, methemoglobin).[83] It should be appreciated that the estimates of hemoglobin-oxygen saturation inferred from nomograms and arterial blood gas analyses are often misinterpreted in this setting; CO-oximetry is necessary to assess the true arterial oxygen saturation (S_{aO_2}) and carboxyhemoglobin saturation (COHb%). It is important to note that pulse oximetry cannot be relied upon to guide this aspect of diagnosis because pulse-oximetry saturation overestimates S_{aO_2} in the presence of COHb.[84] Rapid assessment of cyanide concentration is possible using commercially available kits.

Despite their limitations, arterial blood gas analyses do yield additional essential information in smoke inhalation victims. Hypoxemia and widened alveolar-arterial oxygen gradients are often observed in patients (with or without cutaneous burns) and guide therapy.[85,86] Because delayed hypoxemia often occurs, early measurements of oxygenation at the time of clinical presentation represent an insensitive indicator of injury, and do not accurately predict outcome.[12] Although normal P_{aO_2} does not assure that the patient will have stable respiratory status, the presence of hypoxemia should suggest that significant inhalation injury has occurred and requires further serial assessment.

Numerous focal and diffuse radiographic abnormalities have been observed after smoke inhalation (Fig. 5),[87-89] but the chest radiograph is another relatively insensitive indicator of pulmonary injury. Most admission chest radiographs are normal or reveal (often in retrospect) only mildly accentuated interstitial markings or subsegmental atelectasis. Early pulmonary edema is uncommon, and some may consider it acceptable to reserve chest x-rays for hospitalized patients. However, such studies may also help to exclude other thoracic injuries and provide a baseline for follow-up assessment. As in other patients with acute lung injuries, serial chest radiographs often provide key information otherwise not available. Thus, despite a relatively low diagnostic yield, chest x-rays are advised for every symptomatic patient who has inhaled smoke.[88,89] Routine review of the patient's serial chest films by the respiratory care practitioner and the managing physician provides important perspectives, complements bedside observations, and may add another dimension to care.

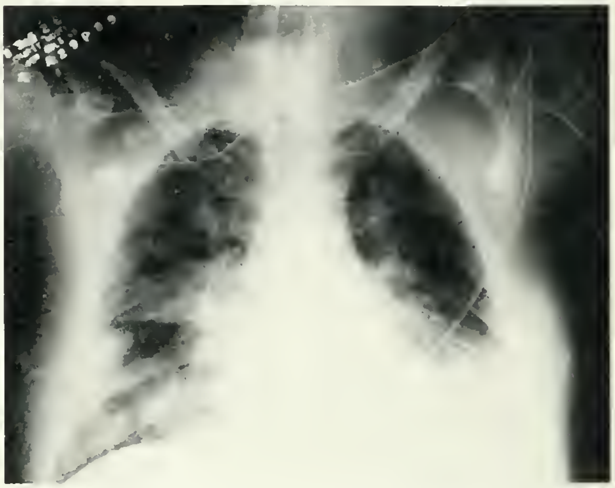

Fig. 5. At one extreme the chest roentgenogram may demonstrate diffuse aveolar disease that is associated with shunt-related hypoxemia as seen in this figure. Most often, however, the admission roentgenogram is normal. (Reprinted, with permission, from Reference 12.)

Direct airway visualization is an essential component of the respiratory assessment,[90-94] and is facilitated by the wide availability of fiberoptic bronchoscopes and nasopharyngoscopes. Several clinical investigations have shown that the prediction of the presence of a compromised upper airway solely on the basis of clinical data is imprecise and may lead to erroneous determinations that either a patient is in no danger or that potentially lethal obstruction is eminent. Endoscopic examination obviates these problems and may help to avoid disastrous errors in management. A spectrum of extramucosal and mucosal abnormalities is seen readily. In general, these findings precede development of abnormalities in arterial blood gases or chest radiographs. In experienced hands and in carefully controlled clinical settings, bronchoscopic complications are unusual in adults. Moreover, serial procedures to assess the upper airway may provide helpful information.[64] Typically, supraglottic damage is more severe than infraglottic injury. Because of its immediate implications for airway support, the upper airway examination is an essential component of the assessment of smoke inhalation victims (Fig. 6). Although true laryngospasm, severe vocal cord edema, and mechanical glottic compromise by secretions and cinders may be observed occasionally, in most patients it is the edema of supraglottic structures that is especially striking. Although complete bronchoscopy is often performed, the implications of inflammation seen during lower airway examination are less clear. An inflamed, soot-covered tracheobronchial tree is seen frequently, but such nonspecific findings have had only variable relationships to concomitant lung parenchymal damage, clinical outcomes, or requirements for mechanical ventilation,[95] and have uncertain implications for management decisions. Nevertheless, at many centers the presence of endobronchial abnormalities is the final criterion establishing that significant inhalation injury has occurred.

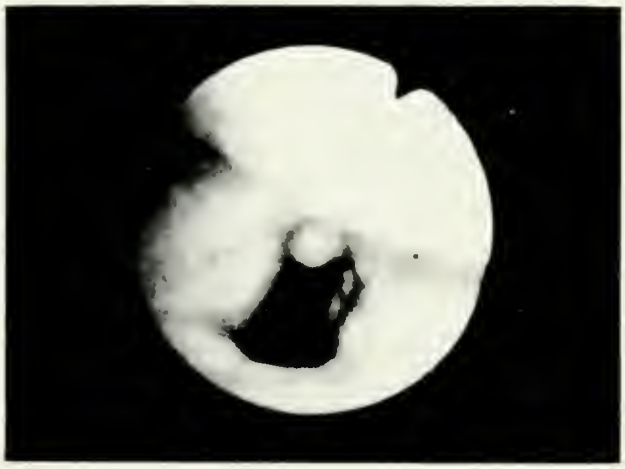

Fig. 6. Upper-airway visualization is essential in symptomatic patients with smoke inhalation. With early injury, edematous supraglottic mucosa is the predominant finding. (Reprinted, with permission, from Reference 12.)

Bronchoscopy has other potentially important clinical and investigative applications in this setting. When chest percussion and postural drainage prove to be either unsuccessful or technically infeasible because of the presence of chest-wall burns, therapeutic bronchoscopy may become necessary to remove copious inspissated secretions.[11] In other patients with upper airway compromise, bronchoscopic techniques may facilitate difficult intubations; the endoscope is used as a stylet over which the endotracheal tube is passed.[96-98] Before extubation, serial bronchoscopic evaluation may contribute to assessment of upper airway status. Preliminary observations have suggested that bronchoalveolar lavage (BAL) can enhance the early diagnosis of smoke inhalation injury and has prognostic value.[99,100] The optimum clinical applications of BAL in this setting are not yet defined; at present, BAL remains a research tool.

Radionuclide scans have also been used to detect inhalation injury.[101-103] Inhomogeneous uptake of tracer, with segmental retention and delayed clearance, may be found and often precedes development of radiographic abnormalities. In some series these findings correlate with mortality from inhalation injury. Intravenous methods (xenon 133) generally are preferred, but are less widely used because of requirements for the radiopharmaceutical, technical support staff, and the predominant roles of other, simpler, and more readily available tests. False-positive results from radionuclide scans can be expected to occur in patients with pre-existing chronic obstructive lung disease or other lung parenchymal disease unrelated to inhalation injury.

Simple pulmonary function tests are perhaps the most underutilized tools in the diagnosis of smoke inhalation, but represent one of the most sensitive means of establishing the diagnosis.[104] Obstructive ventilatory defects occur early and precede radiographic and blood gas abnormalities. Not surprisingly, such physiologic changes are accentuated in the presence of cutaneous burns. Restrictive ventilatory defects accompany large burns, chest-wall involvement, fluid retention, and markedly reduced oncotic pressure.[104-106] Normalization of these physiologic changes tends to parallel clinical improvement. In addition to conventional spirometry (time-volume) and peak flowrate determination (tests that are easily adapted to the emergency situation), flow-volume curves generated with portable equipment are useful (Fig. 7). Not only are inspiratory and expiratory rates reduced, but abnormal patterns of complete flow-volume curves also

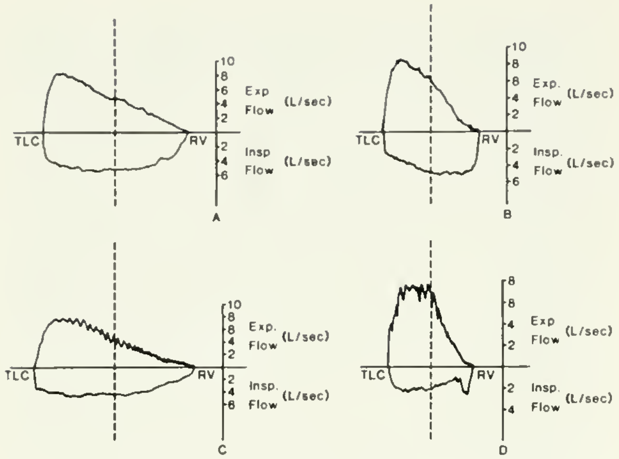

Fig. 7. Abnormal flow-volume curves demonstrating saw-tooth patterns and variable extrathoracic airway obstruction may be early manifestations of smoke inhalation injury. (Reprinted, with permission, from Reference 63.)

may be seen. Patterns of variable and fixed extrathoracic obstruction may be present; their severity has not only correlated with but often has preceded endoscopically visualized upper airway abnormalities.[63,64] The serial, combined use of physiologic testing and fiberoptic nasopharyngoscopy may greatly facilitate accurate identification of patients with acute inhalation injury.[79] Serial charting of peak flow measurements by the respiratory therapist (eg, twice/shift) can provide useful monitoring for nonintubated patients, but no large experience has been reported. Importantly, the presence of completely normal time-volume and flow-volume curves (at the time of the patient's presentation, within hours of exposure to smoke) has an exceedingly high negative-predictive value, and makes it very unlikely that the patient will require endotracheal intubation for inhalation injury.[79]

The use of these diagnostic tests varies considerably with institutional resources and philosophies about management. In general, the respiratory care practitioner has a central role in effectively implementing clinical algorithms for management and monitoring and in dealing with the practical impediments to these plans that occasionally may arise. At our institution we believe that CO-oximetry, visualization of upper airway anatomy, arterial blood gas analysis, and chest roentgenograms are essential to evaluation of all smoke inhalation victims. Abnormalities in results from such studies have obvious implications for management, but normal results do not absolutely exclude clinically significant respiratory damage. Lower airway endoscopy, radionuclide scans, and physiologic measurements complement initial studies and provide definitive and/or helpful information. It should be emphasized that the vulnerability of patients with evolving smoke inhalation injury does not permit the luxury of electively performing all of these tests. Emergency treatment of a patient with clinically obvious respiratory distress must not be deferred pending laboratory confirmation. However, with appropriate advance preparation, a comprehensive evaluation can be performed in a timely way and is easily merged with (and guides) emergency therapy. Clearly, the respiratory care practitioner has a central role in this multidisciplinary approach to patient care.

Managing Patients with Smoke Inhalation Injury

Therapeutic principles of respiratory care that have been validated in other clinical settings have been applied successfully to patients with smoke inhalation. In general, maintenance of a low threshold for suspecting supervening complications, meticulous attention to detail in order to avoid iatrogenic problems, and an active dialogue among all those involved in the patient's care are essential to effective interventions. Maintaining the airway, assuring tissue oxygenation, using mechanical ventilation for hypoventilatory failure, mechanically clearing the airway, and administering selected pharmacologic adjuncts are important aspects of respiratory care (Table 5). Numerous uncertainties about nuances of management make this an important and potentially productive area for clinical investigation.

Airway Issues

As emphasized previously, early elective endotracheal intubation for established or impending UAO is an essential standard of care.[8,58,82] The optimum approach varies with local resources. Because of the high potential for traumatic exacerbation of UAO during intubation attempts (perhaps too often made by the least experienced members of the

Table 5. Therapeutic Priorities in Patients with Inhalation Injury

Respiratory Problem	Response
Asphyxia	Oxygenate and provide antidotes
Upper airway obstruction	Intubate electively early (rarely tracheotomy)
Lower airway obstruction	Institute pulmonary toilet
Mild	Provide bronchodilators and occasionally therapeutic bronchoscopy
Severe (with hypoventilatory failure)	Ventilate mechanically
Parenchymal injury	
Mild	Supplement O_2 (low F_{IO_2} via prongs or mask)
Severe (with hypoxemic failure)	Supplement O_2 (high F_{IO_2} via endotracheal tube)
	Increase lung volume with CPAP or PEEP
	Minimize ongoing alveolocapillary leak (experimental use of antioxidants)
	Adjust volume composition and infusion rates of resuscitative fluid

management team), intubation must be performed by a true expert in airway maintenance.[11] It has been established that tracheotomy,[107-109] particularly in the presence of cutaneous burns, is fraught with hazard, especially due to infection. Accordingly, this procedure is now reserved for situations when an airway cannot be established otherwise or for patients who require prolonged mechanical ventilatory support.

The route of endotracheal intubation varies according to anatomic limitations and the skills of the intubator. Appropriate pulmonary toilet in the presence of large volumes of viscid materials generally requires the largest ETT feasible for a patient, but the presence of pharyngeal edema may markedly limit the size of the tube that can be readily placed. We favor an orotracheal route whenever possible. In the presence of circumoral burns, oral intubations have been associated with lip contractures, and nasotracheal intubation has been recommended. However, this route imposes additional limitations on tube size, and thus affects suctioning and follow-up bronchoscopic examinations. For all these reasons, selection of an appropriate tube remains highly individualized. Rapid sequence intubation is essential in this clinical setting; as noted previously, bronchoscopy may facilitate difficult intubations. It should be appreciated that fatal hyperkalemia has been associated with use of succinylcholine.[110,111] When intubation is performed after the first 8-12 hours, nondepolarizing muscle relaxants are recommended.[12]

Once artificial airways have been placed, they must be managed meticulously to minimize the likelihood of synergistic airway damage produced by the tube and previously injured tracheal mucosa. Close monitoring (and limitation) of cuff pressures becomes a priority of preventive management. Observations in small series suggest that the upper airway and trachea inflamed by smoke may be more vulnerable to superimposed mechanical and tube-cuff trauma.[112-114] Thus, compulsive monitoring (and recording) of ETT position, its stability, and cuff pressures is essential.

Appropriate securing of an ETT (which may have been difficult to place) is a major practical issue. A 'lost' airway too often occurs even in controlled settings of the operating room and burn or intensive care units. The degree of upper-airway edema that is typically present by this time often precludes reintubation. Although emergency tracheotomy is the obvious response to this airway disaster, it is noteworthy that in one recently published series all of the burn patients in whom emergency tracheotomies had been performed for this indication died.[115] In their extensive experience, Clark and co-workers[66] noted a remarkably high frequency of self-extubations, with this problem occurring in 7 of 42 patients with smoke inhalation and surface burns. This frequency was significantly higher than that in burn patients without smoke inhalation injury. In the presence of severe facial and pharyngeal edema, an ETT change (eg, as after a large cuff leak) must be regarded as a major, risky procedure, and must be performed by the most experienced personnel available. Advance preparations for bronchoscopy or tracheotomy should they become necessary are components of this strategy.

Approaches to securing the ETT vary among institutions and with the ingenuity of respiratory care personnel and nursing staff. Surprisingly few data identify the optimum approach in this setting, and this represents one area in which clinical investigation could make considerable impact. In the presence of facial burns, adhesive tape may cause further trauma; moreover, with a typical wet wound, assuring proper adherence is a practical problem. Umbilical tape has been especially favored, but a variety of helmets, halo traction devices, adhesive-backed tapes, twill tapes, Velcro straps, and other devices have been used to secure the airway (and yet avoid traumatizing the patient) in the presence of facial burns and grafts.[116] Despite these measures, self-extubation still occurs too frequently and has been attributed to the technical difficulties of securing the tube, patient agitation, and the requirements for moving the patient during burn wound and other nursing care. As in other situations, particular vigilance during patient transport is mandatory.

Acute upper-airway edema typically resolves during the initial 48 to 72 hours after injury.[12] In patients whose respiratory problems are limited to this anatomic site, early extubation is usually accomplished easily. More prolonged intubation is usually dictated by the patient's needs for mechanical ventilation and/or positive end-expiratory pressure (PEEP) administration for hypoventilatory or hypoxemic respiratory failure, anticipated short-term needs for surgery requiring general anesthesia, persistent neurologic dysfunction, or for any uncertainty about the status of the airway. Most often, when the patient has been intubated solely for upper-airway maintenance, the tube is removed empirically on the third or fourth day. Serial endoscopy may help to assess the status of the airway, but validated criteria for safe extubation have not been established. We have found that visualization of a "glottic chink" with a patent airway lumen immediately adjacent to the ETT suggests (but does not guarantee) that safe extubation is feasible. The value of this strategy, however, has not been established. Inferring resolution of upper-airway edema from measurements of air leak and peak airway pressure when the ETT cuff has been deflated or from tidal flow measurements might be useful, but data derived specifically from burn patients are needed.

As in other settings, extubation is too often underestimated by physicians as a minor event. This attitude courts disaster in patients with smoke inhalation and burns. Appropriate provisions for emergency airway support following a possibly unsuccessful extubation attempt—*every* extubation attempt—are essential. Postextubation stridor may be managed successfully with inhalation of racemic epinephrine, but reintubation is often necessary. A number of adjunctive modalities have been used for acute upper-airway support,[58] but intubation remains the standard of care. Continuous positive airway pressure, administration of helium-oxygen mixtures, or cricothyrotomy with jet ventilation may have roles in persons who cannot readily be reintubated, but there is little documented experience in burn victims. Each of these modalities will place obvious, immediate demands upon the respiratory therapist. Advanced preparations (and appropriate, standardized protocols) are essential to timely responses. In one recent report, the use of helium-oxygen mixtures proved quite useful in reducing postextubation respiratory distress scores among pediatric trauma patients, most of whom had burns.[117] Therapists should recall that proper flowmeters are needed for use of heliox, and that requirements for high oxygen concentration in those patients with severe hypoxemia limits its usefulness.[118] Other aspects of the management of upper-airway complications of thermal injury have been reviewed recently.[58]

Oxygenation and Ventilation

To assure tissue oxygenation, appropriate focus must be placed upon tissue oxygen delivery and not merely on the maintenance of an acceptable P_{aO_2}. The importance of asphyxia due to carbon monoxide and cyanide has been emphasized previously. Accordingly, administration of the highest oxygen concentrations feasible from the time of earliest evaluation in the field assumes the highest priority. This therapeutic opportunity is still too frequently overlooked. The optimum application of hyperbaric therapy for management for carbon monoxide intoxication is addressed elsewhere in this symposium,[119] and has been reviewed recently in a major consensus conference.[120] Although there is considerable agreement that in the presence of elevated COHb levels (eg, > 30%), neurologic dysfunction, and/or underlying cerebrovascular and cardiovascular disease, hyperbaric oxygen is indicated in patients with acute smoke inhalation, considerable controversy exists.[120-123] This management decision presumes that the chamber is immediately available, suitable transportation exists, and appropriate oxygen administration is not deferred or delayed pending arrangements for hyperbaric oxygen. In many instances, smoke inhalation victims have already experienced marked reduction or normalization of COHb levels by the time of their arrival at an emergency room. Whether hyperbaric oxygen is of benefit in reducing the likelihood of delayed neurologic sequelae in these individuals is unclear. The presence of cutaneous burns and requirements for acute fluid resuscitation further influences decisions regarding a 'dive.'

Increased appreciation of cyanide intoxication and its possible synergistic effects with carbon monoxide have led to increasing consideration of empirically administering antidotes to patients at risk.[21-33] It has not been established that this approach is justified for all individuals. Persistent elevations of serum lactate provide another marker of cyanide intoxication following smoke inhalation. In settings in which cyanide is known to be present, elevated serum levels (> 0.2 g/L), or the persistence of altered neurologic status, or metabolic acidosis despite the lowering of elevated COHb levels have been advocated as criteria for antidote use. In one regimen, amyl nitrite is administered initially, followed by intravenous sodium nitrite generating cyanomethemoglobin; subsequently, intravenous sodium thiosulfate is given to produce thiocyanate, which is eliminated.[29,30]

As with other acute respiratory injuries, frequent monitoring of arterial blood gases is essential to guiding modifications of oxygen and ventilator therapy. In the presence of cutaneous burns and related burn shock, tissue delivery may be markedly compromised,[124] and management of the patient's oxygenation is necessarily merged with what are frequently imposing requirements for intravenous fluid resuscitation and hemodynamic support. The optimum approach to a fluid replacement regimen that minimizes the likelihood of alveolar flooding is unclear, and specific institutional approaches are based upon adjustments to standard formulas.[125-127] It is important to note that patients with cutaneous burns and inhalation injury generally receive fluid approximately 35% in excess of the requirements predicted on the basis of their burns alone.[128,129] Although it has been suggested that limitation of this fluid administration might spare the lung, burn mortality is higher with under-resuscitation.[124-131] More data are needed to clarify an optimum approach.

Adjustments of supplemental oxygen and PEEP in patients with hypoxemic respiratory failure must take into consideration changes in hemodynamic status. Although it has been claimed that prophylactic use of PEEP has protective effects in patients with smoke inhalation,[132] this has not been established in a prospective study. PEEP is administered therapeutically as in patients with ARDS due to other etiologies, recruiting lung units and sparing requirements for high concentrations of oxygen.[133,134]

Mechanical ventilation is used for hypoventilatory respiratory failure as in other clinical circumstances. Despite the recent availability of innovative ventilatory techniques, few published reports delineate the role of such techniques in patients with smoke inhalation and/or burns. Many theoretical advantages might be proposed to benefit the diverse lesions of inhalation injury, but proof establishing the superiority of newer technical options in this setting is unavailable. The risks of barotrauma in patients with smoke inhalation favor modes that minimize airway pressures. In a single recent report,[135] high frequency percussive ventilation was associated with a lower occurrence of nosocomial pneumonia and a higher survival than that predicted from historical controls in patients completely managed at one burn center. Unfortunately, no prospective controlled investigation is yet available that documents an optimum approach to mechanical ventilation in this population. Likewise, approaches to weaning patients from the ventilator also tend to vary considerably among institutions and with locally established practices, with no consensus on a best technique.

Mechanical Measures

Mechanical airway clearance is an essential component of respiratory management of patients with smoke inhalation but may be difficult to achieve.[136,137] Chest percussion and postural drainage and an appropriate use of positioning to avoid atelectasis and dependent pneumonias are important contributions to care. Often, however, the presence and distribution of surface burns limits these maneuvers and may lead to a reduced threshold for therapeutic bronchoscopy. Although they are increasingly available, the role of specialty beds in managing these patients with injury has not yet been defined.[138] Sterile foam rubber pads have been used in an effort to protect escharotomy and skin-graft sites during vibratory therapy in patients with surface burns, but attention to appropriate analgesia during these maneuvers is too often overlooked. Although there is no firm documentation of the efficacy of chest physiotherapy in patients with smoke inhalation and/or burns, this modality has an established role in standard regimens.

The risk of exacerbating evolving upper-airway obstruction may prohibit suctioning an unintubated patient. Once again, clear recognition of the danger and objective definition of the status of the upper airway are key. In intubated patients, hemoptysis and exacerbations of hypoxemia may occur. There must also be a heightened awareness of the risk of nosocomial pneumonia and the need for meticulous observation of aseptic technique in an effort to minimize this lethal complication. Usually due to gram-negative bacilli, pneumonia superimposed upon inhalation injury augments mortality by 60%;[14] treatment is especially difficult because of compromised host defenses and the virulence of hospital-acquired pathogens resistant to many drugs. Thus, prevention of this problem altogether is the first-line approach.

Pharmacologic Adjuncts

Surprisingly few data address inhalation therapy in patients with smoke inhalation, and this represents another area in which clinical investigation is greatly needed. Humidification is an accepted component of supportive care. Prophylactic aerosolized antibiotics, aerosolized mucolytics, and expectorants have all been used in patients following smoke inhalation injury; yet, there is little proof of their value in this setting. In patients with tracheobronchial obstruction, inhaled and parenteral bronchodilators have been employed with varying response. Although there are anecdotal observations of improvement, particularly in patients with pre-existing obstructive lung disease, the response to these modalities in smoke inhalation injury has not been established.

Because of the inflammatory nature of acute smoke inhalation, there has been considerable interest in the modification of the inflammatory response and of humoral mediators identified in animal models of smoke inhalation and burns.[139-149] Although there is considerable optimism, sufficient evidence does not exist to support use of antioxidants, free radical scavengers, or other anti-inflammatory agents in humans. Systemic treatment with corticosteroids has, in the past, been advocated for treatment of smoke inhalation.[144-149] The clinical improvement of small numbers of patients with irritant gas injuries (especially due to oxides of nitrogen) had suggested that this approach might help to prevent residual bronchiolitis obliterans. However, prospective investigations in patients with inhalation injury and burns have demonstrated an increased mortality due to infection among steroid-treated patients. In the presence of significant cutaneous burns, steroids should be avoided. Whether short-term therapy might be beneficial in patients with smoke inhalation injury alone is less clear; there are no compelling data to support their general use. Most authorities suggest that steroids be reserved for the treatment of severe bronchospasm unresponsive to other bronchodilator therapy, especially when smoke inhalation has been superimposed upon pre-existing airways disease. Some have advocated short-term steroid administration in patients with severe upper-airway obstruction, particularly after a failed extubation attempt, but objective data are needed to support this application. Although a variety of organic acids may contribute to inhalation injury, there is no indication for the therapeutic lavage of the lung with buffer solutions. Thus, in the absence of definitive therapy that addresses the fundamental respiratory injury, treatment strategies must focus upon the vigorous, albeit nonspecific, support of established physiologic derangements, anticipation (and recognition) of the expected clinical presentations of inhalation injury, and avoidance of the superimposed iatrogenic problems that may frequently complicate the course.

Long-Term Effects

The results of such vigorous acute supportive therapy may be especially gratifying: Although some residual respiratory dysfunction may persist, most patients with isolated smoke inhalation experience remarkably complete resolution of their acute illness.[6] Among chronic sequelae of inhalation injury and its management, however, are several problems, including subclinical airflow obstruction (with airways hyperreactivity), chronic bronchitis, tracheal stenosis (for the most part a complication of airway management), bronchiectasis, bronchial stenosis, bronchiolitis obliterans, pulmonary fibrosis, and tracheobronchial polyps. (Of these problems, tracheal stenosis relates directly to the quality of acute, supportive care and has special implications for respiratory care practitioners.) Each of these problems may present with either acute exacerbations of clinical illness or, more often, the insidious development of respiratory complaints during convalescence. As with the acute injury, a high index of clinical suspicion is necessary for appropriate recognition and intervention.

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Haponik Discussion

Thompson: When I send home kids on oxygen whose parents smoke, I always tell the parents that with 100% oxygen, a polyvinylchloride (PVC) cannula can ignite and give off enough phosgene gas to kill the kid. Do you think I'm lying?

Haponik: This may get the job done, I guess, but I'm unaware of any data to support this suggestion.

Thompson: What's your opinion? Do you need a lot of PVC, a lot of phosgene gas . . .?

Haponik: I don't know. I do agree that the recommendation to stop smoking is the best message the respiratory care practitioner can provide. Educating parents about the proven risks of passive smoke to their children may provide compelling reasons to quit when a smoker may not quit to protect his or her own health.

Rodriguez: Does soot inhalation or inhalation of other hot particulate matter cause particular problems or require different treatment than that of other burns?

Haponik: Generally not, although certainly in some individual patients particulates can create problems. In some patients in addition to insidious upper airway obstruction, you can see mechanical upper airway obstruction from the presence of hot cinders. Sometimes such material in the proximal trachea may cause true focal burns there. I think a major unresolved question relates to the potential for these particulates to increase the residence time of adsorbed gases and potentially result in more severe chemical injury. I think this is speculative in people, but it's a concern. Certainly we don't like the appearance of these secretions, and patients without substantial respiratory injury sometimes expectorate them for 2 to 3 weeks. My main difficulty is in dealing with the volume of these secretions, their potential inspissation, and the problems that present for airway clearance and possible resultant atelectasis.

Durbin: You showed a slide in which you implied that the rate at which one fluid-resuscitated burn patient correlated with the amount of airway edema. I wasn't sure if that was cause and effect or if you were really describing two groups of patients, those who required more rapid versus less rapid resuscitation due to hemodynamic changes.

Haponik: That's precisely what we saw, and those patients tended to have larger body-surface-area burns with a larger resuscitative fluid requirement. Interestingly, those differences in burn size between groups were not statistically significant.[1] It surprised us as well that it was the fluid administration rate and not the burn extent, but I don't think we should overemphasize that because of the small size of the study groups. I think this finding just more or less confirms the long-term clinical observations that during fluid resuscitation one sees very rapid evolution of the upper airway edema. This pace has obvious implications for early intubation.

1. Haponik EF, Meyers DA, Munster AM, Smith PL, Britt EJ, Wise RA, et al. Acute upper airway injury in burn patients: serial changes of flow-volume curves and nasopharyngoscopy. Am Rev Respir Dis 1987;135:360-366.

Thompson: Is the "Rule of 9" still used to estimate body-surface-area burns? As you remember, this is the rapid guide to estimate the extent of a burn:

Hand & neck	9% of BSA
Hand & arm (1)	9% of BSA
Foot & leg (1)	18% of BSA
Anterior trunk	18% of BSA
Posterior trunk	18% of BSA
Perineum	1% of BSA

In children, the proportionately greater surface area of the head makes the calculation inaccurate.

Haponik: Yes it is, but I think that in looking at the whole subject of resuscitation and sorting out the fluid requirements in these patients, we've seen major progress. Cioffi and Pruitt in a recent review[1] have extensively summarized this, and particularly the relationship of surface-area burn to the prediction of fluid requirements.

1. Cioffi WG, Pruitt BA Jr. Resuscitation of the patient with inhalation injury. In: Haponik EF, Munster AM, eds. Respiratory injury: smoke inhalation and burns. New York: McGraw Hill, 1990.

Thompson: Do they still teach that if age plus the body-surface-area burn is greater than 100, the mortality goes up quite a bit?

Haponik: Yes, they do, and I think that's a very realistic way to think about it and to teach our trainees. Interestingly, Heimbach[1] at Seattle and a number of other investigators[2,3] have looked now at the disasterous outcomes of a combination of surface burns in the elderly. Interestingly, the LA$_{50}$, body-surface-area burn causing 50% mortality in the elderly, is only about 5%—a phenomenally low body-surface-area burn in comparison to the much higher LA$_{50}$ (25%) in persons who are less than 60 years old. That experience also showed an increased likelihood of pulmonary edema in those patients relatively early on in the course, suggesting a sensitivity to fluid resuscitation in the presence of pre-existing compromised cardiac function. It may also be related to potential sensitivity to toxic oxygen-radical-mediated injury. The heightened mortality in the elderly is a major current problem.

1. Anous MM, Heimbach DM. Causes of death and predictors in burned patients more than 60 years of age. J Trauma 1986;26:135-139.
2. Manktelow A, Meyer AA, Herzog SR, Peterson HD. Analysis of life expectancy and living status of elderly patients surviving a burn injury. J Trauma 1989;29:203-207.
3. Baux S, Mimoun M, Saade H, Lioret N, Esteve M, Nolland XB, et al. Burns in the elderly. Burns 1989;15:239-240.

Stoller: Could you comment on the role of tracheostomy and the impact of upper airway burns on the initiation of tracheostomy versus leaving the oral endotracheal tube in place?

Haponik: As this group recognizes, the days of placing tracheostomies in these patients early on are fortunately long past. The increased likelihood of nosocomial pneumonia and pulmonary sepsis related to the tracheostomy tube itself, together with the potential risks for tube-related trauma, really prohibit that strategy. We see increasing use of endotracheal (ET) tubes and tubes in place for longer periods of time. As in other clinical settings, it's really problematic as to how long is too long. If there is appropriate monitoring of the tube, use extending out to 2 weeks and then pushing even beyond toward 3 weeks is supported by worthwhile data. Gene Colice has previously reported the problem of tracheostomy and ET-tube-related injuries in these patients.[1] Certainly that's a major long-term sequela for us to avoid. Lund and co-workers[2] also looked at the problem of tracheostomy-related and ET-tube-related injury. I don't have a firm answer for you, in terms of an outer limit for duration of ET intubation, and I think there are many local views on that issue. Who should get an initial tracheostomy? I think it comes back to the indications that we heard earlier this morning: patients in whom establishing an upper airway truly is infeasible otherwise end up comprising the bulk of them.

1. Colice GL, Munster AM, Haponik EF. Tracheal stenosis complicating cutaneous burns: an underestimated problem. Am Rev Respir Dis 1986;134:1315-1318.
2. Lund T, Goodwin CW, McManus WF, Shirani KZ, Stallings RJ, Mason AD Jr, et al. Upper airway sequelae in burn patients requiring endotracheal intubation or tracheostomy. Ann Surg 1985;201:374-382.

Galvin: I realize we're going to hear this tomorrow with the presentation on hyperbaric therapy, but could you comment briefly on what criteria you believe need to be met to implement or utilize hyperbaric treatment?

Haponik: I kept from putting up a slide outlining Eugene Robin's piece in Chest[1] a few years ago: this generated a phenomenal series of letters to the editor. Considering the controversies involved, do I believe hyperbaric oxygen is useful in these individuals? Yes, I do, in a carefully selected subset of patients with carbon monoxide poisoning. My problem is in sorting out which patients. I didn't show a slide outlining relationships of varying carboxyhemoglobin levels to clinical symptoms. There is a lot of variability, and I have difficulty interpreting some of those data. Use of psychometrics, as described in experience from Baltimore,[2] provides additional important information for assessing a patient early on for hyperbaric oxygen. To me the major indication is the presence of neurologic sequelae: The patient who is comatose on presentation or with other profound neurologic dysfunction is in the subset who may benefit from hyperbaric oxygen therapy. That approach presumes that the facility is immediately available; and that the oxygen therapy is going to be appropriately administered while the patient's in transport. Anecdotally, I'm aware of situations in which conventional high-concentration oxygen administration was omitted during all the planning for getting a patient to the hyperbaric facility for the 'dive.' If the victim had been appropriately treated with high concentrations of oxygen from 'the word go,' the problem would have been obviated. Now, that doesn't address the controversial issue of delayed neurologic sequelae. What about that patient who has a low carboxyhemoglobin level at the time of clinical presentation? Does that individual require a dive anyway? It'd be interesting to hear some impressions and, more importantly, the related data. I believe hyperbaric oxygen can be an important adjunct in some individuals, but I think it's a relatively small segment of the group of patients with smoke inhalation, at least from the skewed view of patients whom I see: those patients who have high COHb levels, together with neurologic sequelae, or concomitant cerebrovascular or cardiovascular disease. I hope that comes close to what's suggested tomorrow, but we'll see.

1. Gabb G, Robin ED. Hyperbaric oxygen: a therapy in search of diseases. Chest 1987;92(6):1074-1082.
2. Myers RA, Britten JS. Are arterial blood gases of value in treatment decision for carbon monoxide poisoning? Crit Care Med 1989;17:139-142.

Weaver: I'm speaking tomorrow, and basically that's pretty much what I'm going to say. The bottom line, in my own mind, after treating well over 100 patients with hyperbaric oxygen for carbon monoxide poisoning, is that I don't think I know which patient benefits from it. *Time* is most likely an important variable, based on animal studies with brain lipid peroxidation injury.[1] A prospective blinded trial needs to be performed. I will actually present that argument tomorrow. One comment that I think is pertinent to carboxyhemoglobin level, is that there have been two clinical studies, one by Myers at Baltimore[2] that has clearly demonstrated the carboxyhemoglobin level does not correlate with signs and symptoms, and, in fact, the carboxyhemoglobin half life, which you all who take anesthesia and critical care boards know and have learned to remember, is really not as you learned it in clinical poisoning. That's a manifestation of the laboratory experiment. In clinical poisoning, the carboxyhemoglobin half life is unpredictable,[3] and, in fact, was longer in some patients who received hyperbaric oxygen, in some instances, than in those who did not—presumably because of stores of carbon monoxide other than hemoglobin. So, the bottom line is that the carboxyhemoglobin level only tells you that they were exposed and does not tell you the degree of poisoning. So you need to treat the patient, not the level.

1. Thom SR. Carbon monoxide-mediated brain lipid peroxidation in the rat. J Appl Physiol 1990;68:997-1003.
2. Myers RA, Britten JS. Are arterial blood gases of value in treatment decision for carbon monoxide poisoning? Crit Care Med 1989;17:139-142.
3. Myers RA, Jones DW, Britten JS. Carbon monoxide half-life study. In: Kindwall EP, ed. Proceedings of the 8th International Congress on Hyperbaric Medicine. San Pedro CA: Best Publishing, 1984:263-266.

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Egan's Fundamentals of Respiratory Care, 5th ed, edited by Craig L Scanlan EdD RRT, Charles B Spearman BS RRT, and Richard L Sheldon MD. Hardcover, illustrated, 1,037 pages. St Louis: CV Mosby Co, 1990. $54.95.

I've never really cared for general textbooks. Perhaps in the Age of the Renaissance, when all we had was a smattering of knowledge in every field, such textbooks would have been good reference books. Today, however, if you need a comprehensive source you must turn to texts that specialize in certain aspects of certain fields; this is certainly the case in the field of respiratory care. Nonetheless, there is still a need for general textbooks; for instance, they are useful for first-year students and for those who are preparing for registry exams. (The real problem as I see it is that there are too many general textbooks and not enough specialty textbooks!)

In reviewing **Egan's Fundamentals**, I will simply "walk through" the text (36 chapters organized into 8 sections) and make comments as I go.

Section I: Foundations of Respiratory Care begins with a chapter (not found in the last edition) that attempts to give the reader an understanding of the 'big picture' of health care. This should be helpful for new students (and for certain practitioners who might be afflicted with tunnel-vision). The second chapter deals with the basic functioning of a respiratory care department. Although this chapter is improved in the 5th edition, it still may not be that helpful for most managers because it's too general and it is not really applicable for students or practitioners.

Section II: Scientific Basis for Respiratory Care—Do you know how to pronounce the word "gynecology" correctly? You will if you read Chapter 3 in this section, a new chapter on terms, symbols, and units of measure. The next chapter on physical principles is much improved over the last edition and includes a number of new illustration; however, I would appreciate a better explanation of how some entrainment devices use Bernoulli's principle while others use shearing forces to entrain fluids. The final chapter in Section II (Solutions, Body Fluids, and Electrolytes) devotes less space to atomic structure and calculations and more to explanations—a helpful change in my opinion.

Section III: Applied Life Science—Chapter 6 on respiratory anatomy contains a description of pre- and postnatal development of the respiratory system. In the manner of general textbooks, however, it provides only a cursory look at this fascinating aspect of human development.

Chapter 7, The Cardiovascular System, is nicely done, complete with a lot of new illustrations. The last edition spent a lot of time on EKGs—this edition covers EKGs in a different section and spends more time with descriptions and explanations of the cardiovascular system itself. Ventilation (Chapter 8) emphasizes physiology more than anatomy (unlike the last edition) and appropriately places a greater emphasis on work of breathing. Section III concludes with chapters on gas exchange and transport and acid-base balance as opposed to only one chapter on these subjects in the previous edition. Although some topics are inadequately covered (eg, HbO_2 dissociation curve) and others are overemphasized (eg, Head's paradoxic reflex), these chapters are nicely done.

Section IV: Essentials for Patient Care—All four of the chapters in this section are new. Chapters 12 and 14 in this section (legal/ethical implications and infection control) are similar to the two chapters in Burton's **Respiratory Care** on the same subjects. The other two chapters (11 and 13) are Health Communication and General Patient Care; the former is a bit superfluous, and I believe the latter is too general to be useful to the practitioner.

Section V: The Need for Respiratory Care begins with a chapter on basic cardiopulmonary dysfunction; parts are almost exactly the same (word-for-word) as in the previous edition, but it is improved. The next chapter, Physical Assessment of the Patient (Chapter 16), is new; although it is brief (virtually skipping the importance of patient history), it is still a helpful addition. The next two chapters are my least favorite in the entire book; the chapter on pulmonary function testing is much too brief and the chapter on x-rays is almost useless. When compared with the previous edition, the PFT chapter has been changed only slightly and the x-ray chapter is exactly the same. Why is it that we can't seem to produce a decent monograph on x-ray interpretation? This section ends with a synopsis of cardiopulmonary disease that amounts to little more than a listing of various disorders.

Section VI: Basic Therapeutics begins with pharmacology; besides slimming down a bit and adding one new illustration, this chapter has changed little. Interestingly, there has been a reversal of opinion on aerosolized aminophylline since the last edition, and it seems that the authors are a bit more enthusiastic about saturated solutions of potassium iodide (SSKI) and acetylcysteine than is probably warranted. The other chapters in this section are fairly well done (I did notice that the anode and cathode in a fuel cell analyzer are reversed). I am happy to see that the author of the chapter on humidity therapy leaves out the jet humidifier—current textbooks would be significantly shorter if we'd only get rid of the antiquated information (has anyone used cyclopropane recently to put people to sleep?). Another chapter in this section covers emergency life support, a subject that was not covered in the last edition. For the most part, it contains useful information for both the student and the practitioner.

Section VII: Care of the Critically Ill is a tremendous improvement over the last edition. However, there are still problems: for example, covering neonatal and pediatrics in 40 pages—it can't be done, so why try? In the chapter on mechanical ventilation, the author couldn't decide between "triggering" or "cycling" to describe initiating a breath; however, he did do a nice job of distinguishing between "cycling" and "limiting" (apparently he wasn't overly influenced by McPherson!). I am especially impressed with the explanations for pressure support ventilation (PSV) and mandatory minute ventilation (MMV). The chapter on monitoring has excellent illustrations and a decent explanation of capnography and $S_{\bar{v}O_2}$. It is a little light on pulse oximetry (no pun intended)—but who cares?

Section VIII: New Horizons in Respiratory Care—The first chapter in this section (Health Education and Health Promotion, Chapter 33) was probably included for the sake of completeness, which is an educator's way of saying that it's not terribly important. Two other chapters in this section cover Cardiopulmonary Rehabilitation and Respiratory Home Care; considering that treatment of these subjects in the previous edition was of limited value (in my opinion), these chapters are significantly improved. And, finally, the author of the chapter on computer applications does a nice job of presenting a topic about which most RCPs need to know more.

Despite its shortcomings, **Egan's Fundamentals of Respiratory Care** is still a useful book for students and for those who require a refresher in certain aspects of respiratory care. When it comes to writing a general textbook for respiratory care, I doubt it can be done much better than this one.

Jan K Fields MA RRT
Clinical Coordinator
Muskegon Community College
Muskegon, Michigan

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Pulmonary Pathophysiology—The Essentials, 4th edition, by John B West MD PhD DSc. Soft-cover, illustrated, 219 pages. Baltimore: Williams & Wilkins. 1992. $26.00.

Dr John B West has long been known as an expert in the field of pulmonary physiology. **Pulmonary Pathophysiology—The Essentials**, 4th edition, is intended as a companion text to the fourth edition of **Respiratory Physiology—The Essentials**, a classic discussion of respiratory physiology, which is referenced throughout this text. In the preface, he states that the book "is primarily intended for medical students in their second and subsequent years." His hope is that his book will help bridge the gap between preclinical and clinical disciplines. He also states that it "may prove useful to . . . physicians and paramedical personnel who come into contact with respiratory patients." In this latter group he includes respiratory therapists.

Dr West's attempt to cover pulmonary pathophysiology briefly (193 pages) assumes that the reader is already familiar with normal pulmonary physiology and has been exposed to teaching in pathology. He also apparently assumes that the reader has excellent reading skills and a strong grasp of medical terminology.

The book emphasizes the relationship between structure and function of the diseased lung and is arranged into three sections. Section 1, Lung Function Tests and What They Mean, consists of Chapters 1 (Ventilation), 2 (Gas Exchange), and 3 (Other Tests). Dr West does a good job of explaining the anatomic basis for lung function tests and for interpretation of test results. Chapter 1 covers forced expiratory volumes, flow-volume loops, and single-breath nitrogen tests. Chapter 2 covers blood gas analysis and diffusing capacity using the single-breath carbon monoxide test. Chapter 3 covers tests of static lung volumes, lung elasticity, airways resistance, ventilatory response to CO_2 and hypoxemia, $P_{0.1}$, exercise testing, and regional distributions of ventilation and perfusion.

Section 2, Function of the Diseased Lung, consists of Chapters 4 (Obstructive Diseases), 5 (Restrictive Diseases), 6 (Vascular Diseases), and 7 (Environmental and Other Diseases). I believe this section is far and away the best part of this text. Each chapter focuses on the commonalities of each class of disease with short descriptions of peculiarities in specific diseases. This section is full of diagrams, photographs, and electron micrographs that clearly show the pathologic changes associated with the various classes of disease. The chapter on obstructive disease is one of the clearest, most concise descriptions of the structural changes occurring with emphysema, bronchitis, and asthma that I have seen.

Section 3, Function of the Failing Lung, consists of Chapters 8 (Respiratory Failure), 9 (Oxygen Therapy), and 10 (Mechanical Ventilation). This section deals with physiologic principles of respiratory failure and its chief modes of treatment. In Chapter 8, there is a reasonable listing of the patterns of blood gas values encountered in respiratory failure and descriptions of its associated hypoxemia, hypercapnia, and acidosis. The description of the types of respiratory failure is very brief as is the discussion of their management. Chapter 9 starts well with a good explanation of the responsiveness of various causes of hypoxemia to oxygen therapy and quickly lists factors other than P_{O_2} that influence oxygen delivery. The portion of this chapter dealing with methods of delivery is useless (in fact, the discussion of venturi masks is inaccurate) and should probably be excluded in future editions. CO_2 retention and absorption atelectasis are well covered under hazards of oxygen therapy, but oxygen toxicity and retrolental fibroplasia are superficially discussed. In Chapter 10, a great deal of space is wasted on an inadequate discussion of the technical aspects of mechanical ventilators and an incomplete discussion of modes of ventilation (limited to IPPV, PEEP, CPAP, IMV, and high frequency ventilation); however, this chapter is saved by the discussion of the physiologic effects of mechanical ventilation. Miscellaneous hazards like pneumothorax, interstitial emphysema, infection, arrhythmias, and gastrointestinal bleeding are covered in 10 sentences.

The Appendix lists some common symbols and gives a chart for calculating normal pulmonary function values. A bibliography and related texts is included to assist the reader in locating more detailed information on topics of interest. The book concludes with a list of questions and answers for each chapter so the reader can test his comprehension of the material. These questions are the same as in the third edition except that a few have been added.

This is a generally well-written text by a knowledgeable author. The figures and illustrations tend to be clear and support the points well, but some of the figures depicting distribution of blood flow and ventilation in various diseases are confusing (eg, Figs. 6.9 and 10.4). There are remarkably few typographical errors. This book would be of value to someone with strong reading skills, a thorough understanding of medical terminology, and a good background in respiratory physiology.

This fourth edition is improved over the third edition and shows revision throughout the text. Dr West has included up-to-date information on the pathophysiology of the various diseases and has in general improved the readability of the text.

The main strength of this text lies in its discussion of the pathophysiology of the various diseases. Its main weakness is that it covers too much material in too short a space. The chapters dealing with oxygen therapy and mechanical ventilators detract from an otherwise fine book.

In conclusion, I feel that this book meets its intended goal. I also feel that it could serve as a useful review of pulmonary pathophysiology for practicing respiratory care personnel. However, I do not feel that this text is appropriate for students in a respiratory therapy program because it requires too high a comprehension level and assumes too much background knowledge.

Arthur A Taft MHS RRT
Director of Clinical Education
Respiratory Therapy Program
Medical College of Georgia
Augusta, Georgia

Allen & Handburys Athletic Drug Reference (pocketbook edition) is available for $4.95 plus $2.75 postage and handling from Clean Data Inc, PO Box 14183, Durham NC 27709-4183. Checks, money orders, and Mastercard and Visa accepted.

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See July RESPIRATORY CARE for more information.

Editor's Note: This table was omitted from "Sighs: Wasted Breath or Breath of Fresh Air?" published in Kittredge's Corner in May Issue (Respir Care 1992;37:462-468). We regret the error. We suggest you detach this page and put with the article itself.

Table 1 Sigh Capabilities of Ventilators

Ventilator	Sighs	Modes	Frequency (per hour)	Volume	Volume Range (L)	Maximum Inspiratory Time	Multiple Sighs	Separate High-Pressure Alarm (cm H_2O)	Manual Sigh
Bear 1, 2, 3	Yes	CMV, AMV*	2-60	Adjustable	0.15-3.0	No	1, 2, or 3	100, 120	Yes
Bear 5	Yes	All except time cycle	2-60	Adjustable	0.065-3.0	No	1, 2, or 3	140	Yes
Bird 6400ST & 8400ST	Yes	All	Every 100 breaths	1.5 × set V_T	0.075-3.0	No	1	1.5 × set alarm up to 140	No
Emerson	No	—	—	—	—	—	—	—	—
Hamilton Veolar & Amadeus	Yes	All except PCV	Every 100 breaths	1.5 × set V_T	0.075-3.0	No	1	set alarm + 10	No
Infrasonics AdultStar	Yes	All	1-20	0.1-2.5 Adjustable	0.1-2.5	No	1, 2, 3	1.5 × set alarm up to 120	Yes
Impact Uni-Vent 750	Yes	All	Every 100 breaths or every 7 min	1.5 × set V_T	0.15-3.0	3.0	1	No	No
Newport E-100i	No	—	—	—	—	—	—	—	—
Newport Breeze	Yes	AMV	Every 100 breaths	1.5 × V_T	0.45-3.75	I:E 4:1	1	No	No
Newport Wave	Yes	SIMV or AMV	Every 100 breaths	1.5 × V_T	0.45-3.75	I:E 3:1	1	No	No
Ohmeda Advent	Yes	AMV, SIMV, MMV	Every 100 breaths	1.5 × V_T	0.1-3.0	4.0	1	No	Yes
PPG IRISA	No	—	—	—	—	—	—	—	—
Puritan-Bennett MA-1	Yes	All	4-15	Adjustable	0.1-2.2	No	1, 2, 3	20-80	Yes
Puritan-Bennett 7200ae	Yes	All	1-15	Adjustable	0.1-2.5	No	1, 2, 3	10-120	Yes
Siemens 900C	Yes	Volume control	Every 100 breaths	2 × V_T	0.1-3.0	I:E 4:1	1	No	No
Siemens 300	No	—	—	—	—	—	—	—	—

---

*CMV = Control mechanical ventilation; AMV = assist-control mechanical ventilation; PCV = pressure-control mechanical ventilation; SIMV = synchronous intermittent mechanical ventilation; MMV = mandatory minute ventilation; and V_T = tidal volume.

Listening to the Chest in the 1930s*

Auscultation of the Lungs

In listening to the lungs, we have two methods at our disposal, immediate auscultation which consists in placing the ear directly against the chest wall, and mediate auscultation which consists in listening through a stethoscope. While the mediate method is the method universally employed, the student should become familiar with the immediate method as well. The occasion may arise when the physician through unforeseen circumstances does not have his stethoscope with him. In examining babies or children the appearance of the stethoscope may excite the fear of the patient who does not object to the examiner placing his ear against the chest. Again, just as the examiner always has his fingers with him for percussion, so he always has his ears for auscultation.

Stethoscopes

The earliest type of stethoscope was the monaural stethoscope devised by Laennec. This stethoscope, usually made of wood, is 1 foot in length, has a funnel-shaped enlargement on the end which is placed on the chest, and a flat flange on the end which is applied to the ear (Fig. 200). This type of stethoscope, while somewhat of a curiosity in this country, is still extensively employed in European clinics, where physicians still champion its superiority and point to its use for more than a century (Fig. 201). There is no question of its value in the hands of those familiar with it. Most, if not all, of the great discoveries in auscultation have been made with this instrument. Yet it is cer-

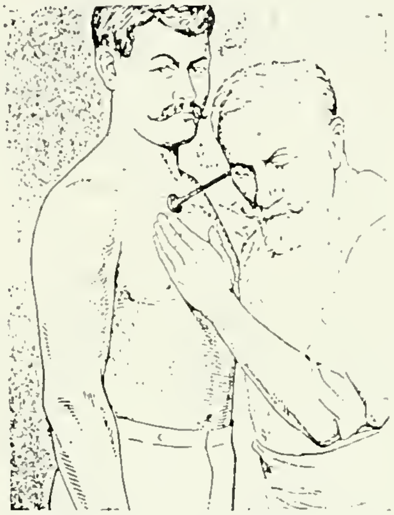

Fig. 200. Application of monaural stethoscope.

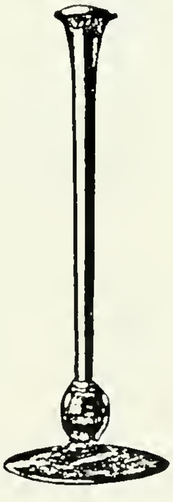

Fig. 201. Monaural stethoscope.

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* These excerpts are from the book Physical Diagnosis, 3rd edition, by Ralph H Major MD, Professor of Medicine at The University of Kansas. The publisher was WB Saunders Company of Philadelphia. The book first appeared in 1937; these excerpts were from the edition printed in 1948. Larry Boller PhD RRT, Director of Allied Services at The Monroe Clinic, Monroe, Wisconsin, discovered this material and made it available to RESPIRATORY CARE. Reprinted with the permission of WB Saunders Company.

tainly less convenient to use than the binaural type. Thoroughly to explore various regions of the chest with this stethoscope sometimes requires an almost acrobatic agility on the part of the examiner. The American physician usually complains that with the monaural stethoscope he hears too little, to which his Continental colleague retorts that with a binaural stethoscope he hears too much . . ..

Hippocratic Succussion

This classical sign was first described by Hippocrates* in empyema. "Set the patient on a stool, which is not wobbly, someone should hold his hands, then shake him by the shoulders and listen to see on which side a noise is heard." (Fig. 217).

Laennec was the first to recognize that Hippocratic succussion is due to the presence of both air and fluid in the chest and to point out, that even a large amount of fluid in the chest produces no splashing sound, when the patient is shaken, unless there is also air present. Air is necessary to produce a splashing sound. A bottle full of water can be shaken violently without the production of a splashing sound, a bottle half-filled with water gives a splashing sound when agitated violently.

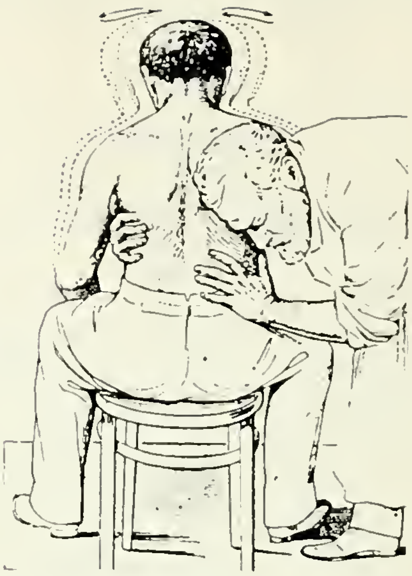

Fig. 217. Hippocratic succussion.

This splashing sound is often heard by the patient when he walks or climbs stairs. At other times he feels the displacement of the fluid caused by change in posture. Morgagni described a patient who "said that when she moved her chest she felt a splashing within."

The succussion is best elicited by following the ancient method of Hippocrates, shaking the patient by the shoulders and listening with the ear or stethoscope against the chest. The splashing sound caused by disturbing the level of fluid is easily heard.

---

*Editor's Note: Hippocrates lived around 600 BC.

Editor's note: This Historical Notes article was originally published in March 1992; however, due to a production error, the figures were omitted.

We regret the error.

Not-for-profit organizations are offered a free advertisement of up to eight lines to appear, on a space-available basis, in Calendar of Events in RESPIRATORY CARE. Ads for other meetings are priced at $5.50 per line and require an insertion order. Deadline is the 20th of the month two months preceding the month you wish the ad to run. Submit copy and insertion orders to: Calendar of Events, RESPIRATORY CARE, 11030 Ables Lane • Dallas TX 75229-4593.

Calendar of Events

AARC & AFFILIATES

June 16-18 in Scottsdale, Arizona. The ASRC holds its Annual Seminar, "Summer Mirage '92," at the Wyndham Paradise Valley Resort. Featured topics include RSV, COPD: Past and Future, New Advances in Cardiovascular Care, and Waveform Analysis for Appropriate Ventilator Adjustment. AARC Director of Government Affairs Cheryl Brown will give a special presentation on home health issues and CLIA '88. Also featured are more than 60 vendor exhibits, Sputum Bowl competitions, and two nights of entertainment. Contact Sue Meckert, PO Box 34344, Phoenix AZ 85067-4344; (602) 285-3250.

June 25-27 in San Diego, California. The CSRC holds its 24th Annual Convention at the Sheraton Harbor Island Hotel. The meeting features over 100 exhibitors, Sputum Bowl competition, and a dance. Contact John Bigler (805) 298-4010.

July 9 AARC Videoconference. The AARC, in conjunction with VHA Satellite Network, presents "Pulmonary Rehabilitation," one in a series of live satellite videoconferences titled "Professor's Rounds in Respiratory Care." Featured presenters are Drs Barry Make and David J Pierson. Site registration for entire staff is $245 for AARC members. Call (214) 830-0061.

July 15-17 in San Antonio, Texas. The TSRC presents its Annual Convention and Trade Show, "Kaleidoscope of Quality." Featured speakers include Sam Giordano, Dean Hess, Robert Demers, Robert Kacmarek, and Neil MacIntyre. Social events include a barbecue, 3-K fun run, dances, and an awards ceremony. Contact the TSRC Executive Office at (214) 680-2454.

July 24-26 in Naples, Florida. The AARC's Summer Forum, featuring education and management programs, held at the Registry Resort. For details, refer to the special Summer Forum Program in the April *AARC Times* or call (214) 243-2272.

August 27 in Sacramento, California. The CSRC, Chapter 11, presents its Annual Seminar, "Tools of the Trade," in conjunction with the California State Fair. The seminar, admission to the fair, dinner at the fair's VIP tent, and California wine tasting are included in the admission price. Contact Marie Kearney (916) 441-7626 or Mark Goldstein (916) 933-3469.

October 14 in Auckland, New Zealand. The NZSRC presents its Annual Scientific Meeting at the Aotea Centre. The meeting precedes the Australian-New Zealand Intensive Care Society Conference. Contact Graeme A'Court, PO Box 10148, Balmoral, Auckland, New Zealand. (643) 640640.

OTHER MEETINGS

June 18-19 in Des Moines, Iowa. The Respiratory Care Departments of Iowa Methodist Medical Center and Mercy Hospital Medical Center present the 3rd Annual Respiratory Seminar. Topics include legal issues, ventilator management, post-anesthesia concerns, and updates on airway clearance. Contact Delite Lester, Mercy Hospital Medical Center. (515) 247-4197.

July 29-31 in Salt Lake City, Utah. The Rocky Mountain Center for Occupational and Environmental Health presents "Training for Pulmonary Function Testing," a 2 1/2 day course for personnel involved in the performance and interpretation of screening spirometry. Call (801) 581-5710, or write Program Coordinator, Rocky Mountain Center for Occupational and Environmental Health, Building 512, University of Utah, Salt Lake City UT 84112.

September 6-13 on a Caribbean Cruise. Dream Cruises presents its 6th Annual Labor Day Cruise, "Sea No Evil." Ports of call include Miami, Nassau, Blue Island, San Juan, St John, and St Thomas. Starting price with air is $925 per person/double occupancy. Contact Kathy Kearney at 10882 La Dona Ave, Garden Grove CA 92640. (800) 462-3628 or (714) 636-6660.

Notices

Notices of competitions, scholarships, fellowships, examination dates, new educational programs, and the like will be listed here free of charge. Items for the Notices section must reach the Journal 60 days before the desired month of publication (January 1 for the March issue, February 1 for the April issue, etc). Include all pertinent information and mail notices to RESPIRATORY CARE Notices Dept, 11030 Ables Lane, Dallas TX 75229-4593.

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I. Type of Instn/Practice
1 ☐ Hosp ≥ 500 or more beds
2 ☐ Hosp 300 to 499 beds
3 ☐ Hosp 200 to 299 beds
4 ☐ Hosp 100 to 199 beds
5 ☐ Hosp <100 or less bedS
6 ☐ Skilled Nursing Facility
7 ☐ Home Care Practice
8 ☐ School
II. Department
A ☐ Respiratory Therapy
B. ☐ Cardiopulmonary
C. ☐ Anesthesia Service
D. ☐ Emergency Dept
III. Specialty
1 ☐ Clinical Practice
2 ☐ Perinatal Pediatrics
3 ☐ Critical Care
4 ☐ Clinical Research
5 ☐ Pulmonary Function Lab
6 ☐ Home Care/Rehab
7 ☐ Education
8 ☐ Management
IV. Position
A. ☐ Dept Head
B ☐ Chief Therapist
C ☐ Supervisor
D. ☐ Staff Technician
E ☐ Staff Therapist
F ☐ Educator
G ☐ Medical Director
H ☐ Anesthesiologist
I ☐ Pulmonologist
J ☐ Other MD
K ☐ Nurse
V Are you a member of the AARC?
1 ☐ Yes

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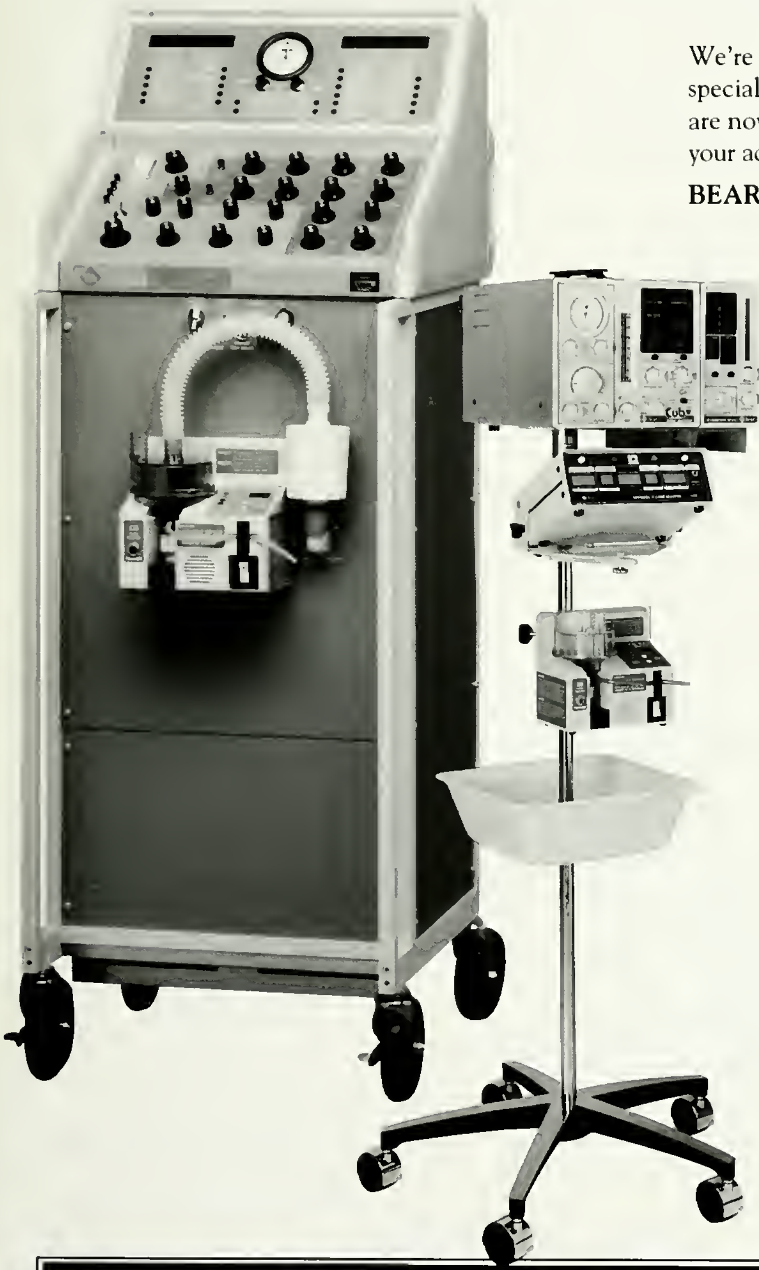

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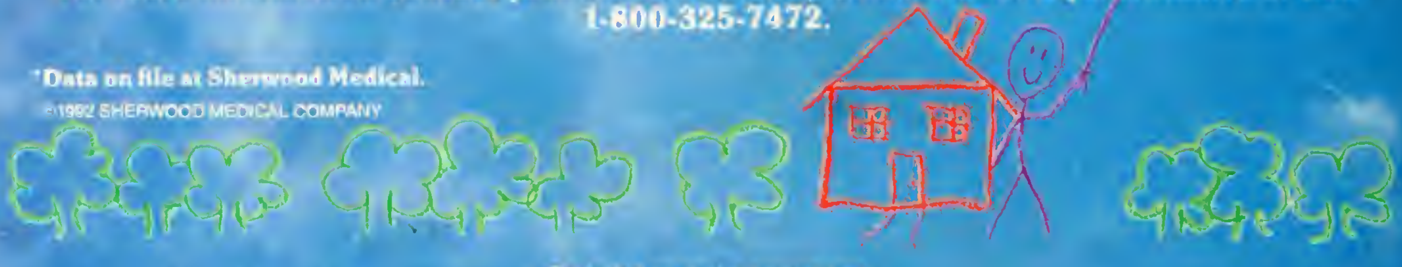

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